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# Original Communications

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## CONSTRICTIVE PERICARDITIS AND CONSTRICTIVE PLEURITIS TREATED BY PERICARDIECTOMY AND PULMONARY DECORTICATION

### REPORT OF A CASE

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CONSTRICTION of the pericardium and pleura represents an unfortunate paradox in which the process of healing creates a disability greater than the original disorder. Since the function of both heart and lung is dependent upon expansion and contraction, disability is proportionate to the area and the degree of inelasticity of the constricting scar.

Constrictive pleuritis is the more common. Although it limits ventilation and, therefore, may be disabling, it is in itself rarely fatal. Constrictive pericarditis is less frequent; if severe, it is disabling and may progress to a fatal conclusion. Should these two constrictions occur simultaneously, the chances for a successful pericardiectomy will be greater with recognition and remedy of the pleuritis.

The only recorded recognition of combined constrictive disability of heart and lung was published by Burwell and Ayer[1] in 1941. The disease was of tuberculous etiology. A pericardiectomy relieved the associated circulatory effects, but death resulted from extreme ventilatory insufficiency produced by the pulmonary immobility.

There are four reasons for our presentation of another case of this combined constrictive disability: (1) unusual etiology, (2) preoperative recognition, (3)

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simultaneous and successful treatment, and (4) a new operative approach for pericardiectomy.

## CASE REPORT

T. J. F., a 50-year-old white unemployed painter was admitted to the New England Deaconess Hospital on July 5, 1950, with complaints of cough, dyspnea, and progressive edema. In February, 1945, this patient received a nonpenetrating injury to the chest when he was crushed against a concrete pillbox by a truck. He was hospitalized for many months with bilateral numerous rib fractures and hemothoraces. He was treated with rib traction, body cast, and bilateral closed catheter drainage. Since the injury, he had had gradually increasing exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. Repeated respiratory infections, characterized by severe dyspnea and cough, had necessitated hospitalization for oxygen, bronchodilator, and antibiotic therapy. At least one of these episodes was accompanied by substernal pain. Progressive edema and ascites, unsuccessfully treated with digitalis and diuretics, had been present for six months prior to admission.

*Physical Examination.*—The patient was a moderately dyspneic and orthopneic man with visibly distended neck veins and engorged retinal veins. The chest was held in an inspiratory position, and the anteroposterior diameter was definitely increased. The left side moved considerably more than the right, and the right lower chest almost not at all. Dullness and decreased breath sounds were present at the right base, and there were fine râles at both bases. The heart was not grossly enlarged; its rhythm was regular; and its sounds were distant and unaccompanied by murmurs. There was a marked *pulsus paradoxicus* with a fall in systolic blood pressure of 15 mm. Hg on inspiration. There was extensive edema, most marked in the lower half of the body, but present also in the arms. Liver dullness extended two fingerbreadths below the right costal margin.

*Laboratory Findings.*—Hemoglobin was 15 mg. per cent, with a hematocrit of 47 per cent. Electrocardiogram showed low to inverted T waves and low EMF of QRS in all leads. Chest film showed numerous old rib fractures with markedly thickened pleura over all surfaces of the lungs and obliteration of the right costophrenic angle (Fig. 1). At fluoroscopy, the right diaphragm moved only 1 cm., the left, 4 cm. The heart was not enlarged, but amplitude of the heartbeat was much reduced on all borders. No pericardial calcification was seen. Venous pressure in both antecubital and femoral veins was 350 mm. of saline.

*Course.*—The preoperative pulmonary function studies were done on July 15, 1950, and cardiac catheterization on July 17, 1950. Decortication of the left lung and the heart was done under local anesthesia with the patient in the prone position on July 26, 1950. The pleural space was found to be completely obliterated, and the scar measured 2 to 3 mm. in thickness. The pulmonary decortication was accomplished with ease, and complete expansion of the lung was observed. There was no evidence by inspection or palpation of parenchymal pulmonary disease. The cardiac pulsations were markedly diminished by a thick, fibrotic layer of tissues, largely replacing the pericardium and densely adherent to the myocardium. This thickened pericardium measured 4 to 5 mm. in thickness. No calcification was observed. The constricting pericardial membrane was removed from the entire surface of the left ventricle and from a portion of the right ventricle and of the left auricle. Following this the cardiac pulsations appeared normal. During the actual removal of the cicatrix, cardiac arrhythmias developed on several occasions, but returned to normal following rest from the manipulation. A short episode of cardiac standstill responded quickly and favorably to cardiac massage. The phrenic nerve was carefully dissected from the cicatrix, and at completion of the surgical procedure the diaphragmatic motions on the left were noted to be normal. The venous pressure was 180 mm. of saline three weeks postoperatively, and 140 mm. of saline five months later. Decortication of the right lung was performed on Dec. 11, 1950. The pleural space was completely obliterated, and the thickened scarred membrane was more dense and adherent than that encountered in the left hemithorax. This membrane measured 2 to 4 mm. in thickness. Following decortication, there was pulmonary expansion. No gross evidence of pulmonary parenchymal disease was observed. Inspection of the heart and pericardium on the right side revealed pulsations to be normal, and there was no evidence of any constrictive phenomenon. The postoperative course was uncomplicated. The pulmonary function studies were repeated on March 17, 1951, and cardiac catheterization on March 19, 1951.

*Etiology.*—The cause of the pericardial and pleural cicatrix in this case is of particular interest. There was no microscopic evidence of tuberculosis in any of the specimens, and the pleural membrane had none of the gross characteristics of the peel resulting from empyema, tuberculous or otherwise. It is known that the patient had bilateral hemothoraces following his crushing injury; however, it is highly unlikely these ever became infected since open drainage was never necessary, and since at pulmonary decortication no empyema pocket was found on either side. During his illness, there was no history suggestive of pericardial effusion or purulent pericarditis. We believe that this combined constrictive disability was the late result of a sterile hemothorax and hemopericardium. We have been unable to find a reported, well-substantiated case of constrictive pericarditis resulting from injury uncomplicated by infection. Warburg[2] has included in his monograph on nonpenetrating injuries of the heart a case of chest injury followed by a typical Pick's syndrome with pericardial calcification demonstrated by roentgenogram. No post-mortem examination was reported, however, and incidental tuberculosis in his patient cannot be excluded. A case reported by White and Glandy[3] had proved tuberculous constrictive pericarditis following a bloody pericardial effusion due to injury.

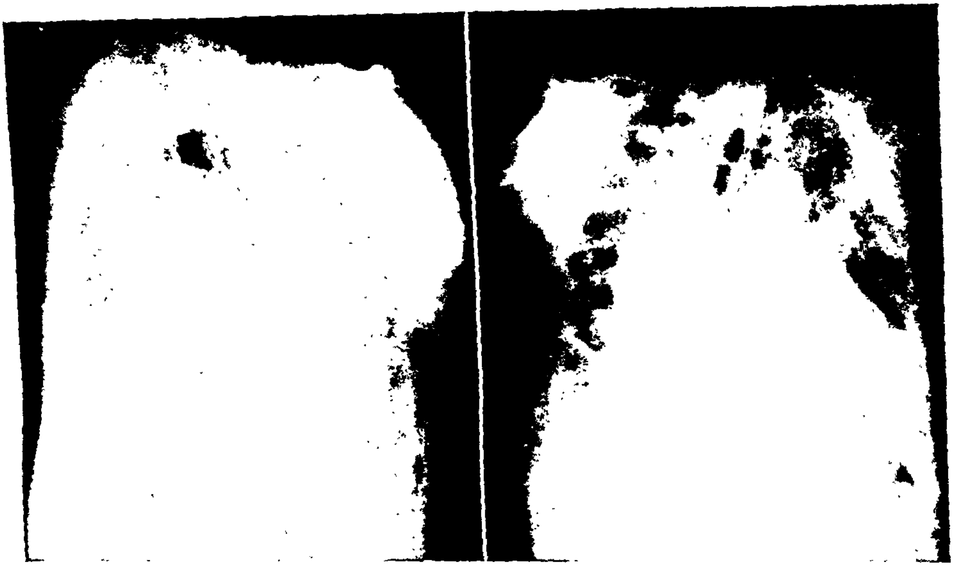

Fig. 1.—Preoperative oblique roentgenogram. This demonstrates the bilaterally thickened pleural membrane.

*Cardiac Catheterization.*—This patient was subjected to cardiac catheterization preoperatively and eight months after his pericardiectomy. Before presenting the actual pressure values obtained in our case, it may be worth while to review the normal pressure-flow relationships in both systemic and pulmonary circuits and the various concepts of pressure alteration produced by constrictive pericarditis. The normal values for pressures within the heart and great vessels are presented in Fig. 2. In Fig. 3 are illustrated the possible sites at which obstruction to the circulation might be caused by constrictive pericarditis as postulated by various authors.[4-11] For purposes of illustration, hypothetical pressures which would have to be obtained with each site of obstruction are also noted. In our experience, the pressure relationships indicated in the three hypothetical cases have never been encountered.

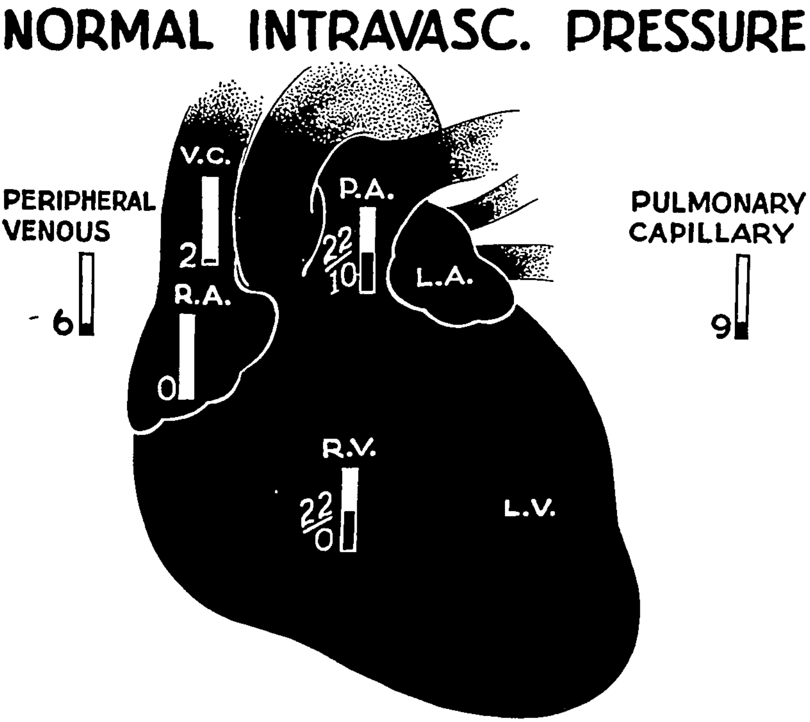

Fig. 2.

The normal values and the pre- and postoperative values of this patient are listed in Table I. It is to be noted that the "pulmonary capillary" pressure, pulmonary arterial diastolic pressure and right ventricular diastolic pressure are almost identical and markedly elevated preoperatively in our patient. These pressure changes are characteristic of constriction of the left ventricle.[11] The only recorded catheterization data in constrictive pericarditis have indicated that constriction of the left ventricle was present in every case (9 and 11); therefore, unless catheterization data clearly indicate in any individual case that marked constriction is present at any other site, it is the surgeon's responsibility to remove adequately the constricting scar over the left ventricle. Postoperative catheterization data in this patient show an increase in cardiac output (expressed as liters per minute per 100 c.c. of oxygen consumption) and in

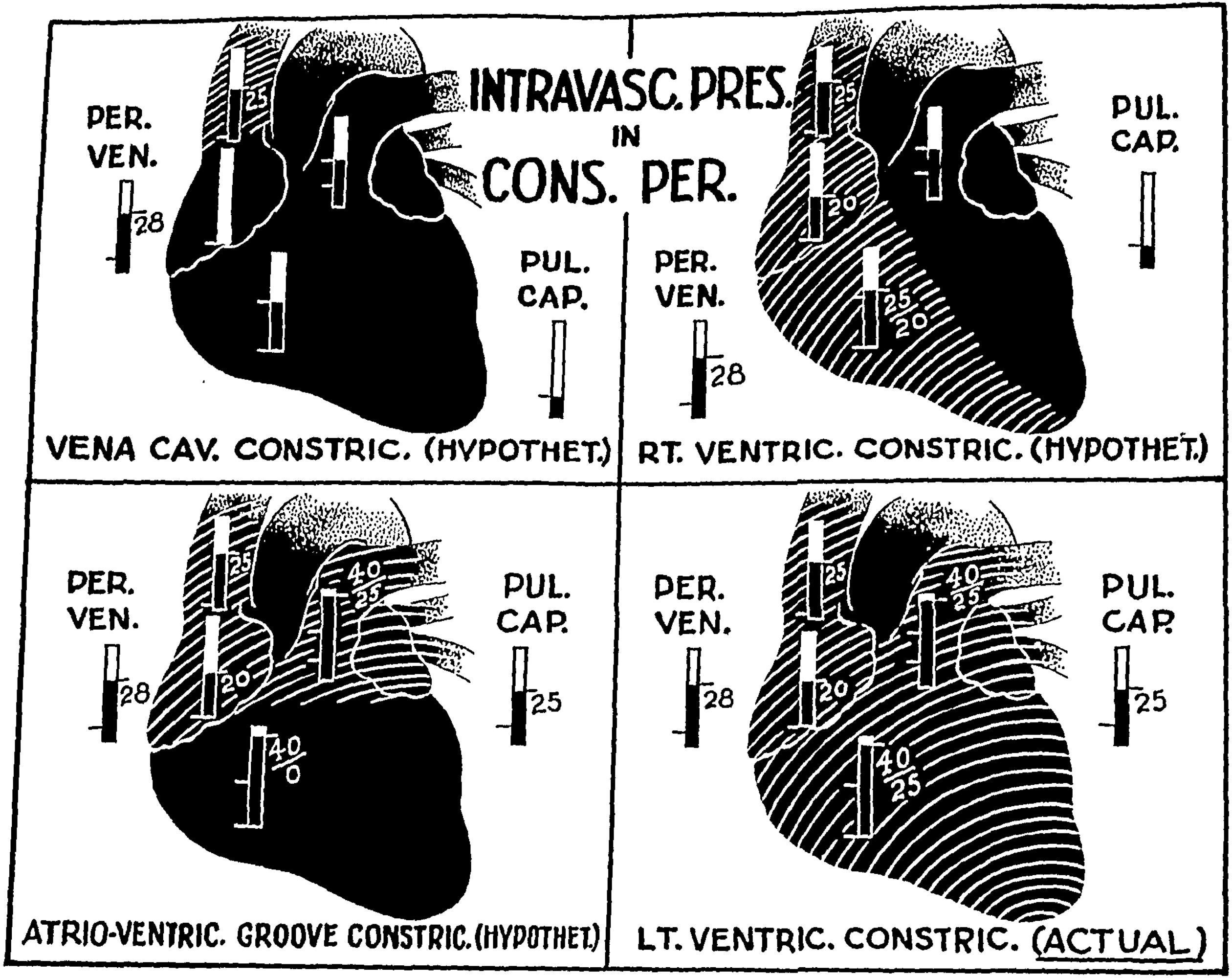

Fig. 3.—Diagram indicating intravascular pressures in constrictive pericarditis. The area of the curved white line represents elevated intravascular pressures due to constrictive pericarditis.

stroke index as well as a return of all pressures to near the normal values. The residual elevation of "pulmonary capillary" pressure may be attributable to left ventricular failure due to the myocardial atrophy seen routinely after pericardiectomy[12] or due to fibrosis of the myocardium.

TABLE I. CARDIAC CATHETERIZATION DATA

T. J. F. MALE, AGE 50 CONSTRICTIVE PERICARDITIS

| | | NORMAL RANGE | PREOPERATIVE *JULY 17, 1950* | 8 MONTHS POSTOPERATIVE *MARCH 19, 1951* |
|---|---|---|---|---|
| Height (cm.) | | | 165.5 | 165.5 |
| Weight (Kg.) | | | 71.6 | 63.6 |
| Body surface (area M$^2$) | | | 1.77 | 1.71 |
| Intravascular pressures mm. Hg | | | | |
| Peripheral arterial | (S/D) | 120/80 | 134/82 | 155/81 |
| Peripheral arterial | (mean) | 100 | 95 | 118 |
| "Pulmonary capillary" | (mean) | 9 | 27 | 18 |
| Pulmonary artery | (S/D) | 22/10 | 45/29 | 34/12 |
| Pulmonary artery | (mean) | 12 | 34 | 22 |
| Right ventricular | (S/D) | 22/5/0 | 46/25/15 | 33/12/3 |
| Right atrial | (mean) | 0 | 22 | 7 |
| Vena caval | (mean) | 2 | 25 | 8 |
| Peripheral venous | (mean) | 6 | 27 | 8 |
| Cardiac output | | | | |
| Arterial oxygen content | (Vol. per cent) | | 19.25 | 19.45 |
| Venous oxygen content | (Vol. per cent) | | 14.45 | 15.38 |
| *Arterial oxygen saturation* | (*per cent*) | | 96 | 98 |
| A-V difference | (c.c./L.) | | 48 | 41 |
| Oxygen consumption | (c.c./min.) | | 222 | 319 |
| Oxygen consumption | (c.c./min./M$^2$) | | 125 | 187 |
| Ventilation | (L./min.) | | 7.8 | 12.0 |
| Cardiac output | (L./min.) | | 4.6 | 8.0 |
| Cardiac output | (L./min./100 c.c. 0$^2$) | | 2.1 | 2.5 |
| Cardiac index | (L./min./M$^2$) | | 2.6 | 4.7 |
| Pulse rate | (beats/min.) | | 82 | 80 |
| Stroke volume | (c.c./beat) | | 56 | 100 |
| Stroke index | (c.c./beat/M$^2$) | | 32 | 58 |

TABLE II. VENTILATORY FUNCTION STUDIES

T. J. F. MALE, AGE 50 CONSTRICTIVE PLEURITIS AND PERICARDITIS

| | | PREOPERATIVE JULY 15, 1950 | *POST LEFT* DECORTICATION & PERICARDECTOMY DEC. 5, 1950 | POST RIGHT DECORTICATION MARCH 17, 1951 |
|---|---|---|---|---|
| Respiratory rate/min. | | 16 | 13 | 20 |
| Tidal Air at Rest | (L.) | 0.457 | 0.682 | 0.488 |
| Resting Minute Volume | (L./min.) | 7.30 | 8.52 | 10.05 |
| Expiratory Capacity | (L.) | 0.478 | 0.870 | 0.880 |
| Inspiratory Capacity | (L.) | 0.870 | 1.160 | 0.946 |
| Vital Capacity | (L.) | 1.190* | 1.960<br>1.940* | 1.805<br>1.715* |
| Maximum Breathing Capacity | (L./min.) | 32.6* | 72.5<br>53.0* | 74.2<br>55.0* |
| Resting Ventilatory Reserve | (per cent) | 77.6* | 88.3<br>84.0* | 86.6<br>82.0* |
| Capacity Ratio $\left(\frac{MBC}{VC}\right)$ | | 27.4* | 37.0<br>27.4* | 41.0<br>32.0* |

*Values obtained with valves and soda lime canister in ventilometer offering significant resistance.

*Ventilatory Function Study.*—Pulmonary decortication is now a standard procedure for incarcerated lung in chronic hemothorax, nonexpandable pneumothorax, and chronic empyema.[13, 14] Respiratory function studies in patients subjected to pulmonary decortication have been reported by several authors.[15-18] This experience indicates that excellent restoration of ventilation and gas exchange can be expected after decortication when the underlying lung parenchyma is not extensively diseased and when the incarceration has not been of excessive duration. Little benefit has been derived from decortication in most cases of mixed tuberculous empyema and in some of these decortication has made even worse the ventilation-perfusion relationship in a diseased lung by increasing its ventilation without increasing its oxygen uptake.[17, 18]

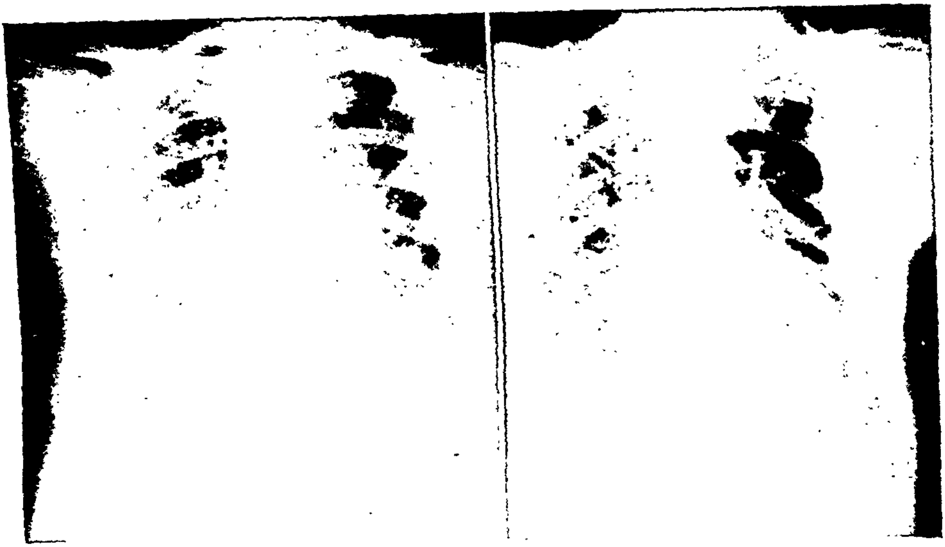

Fig. 4.—Preoperative posteroanterior roentgenograms of the chest (left). The numerous rib fractures and bony bridges are noted. The same projection postoperatively (right). Note the altered cardiac silhouette following the release from the pericardial constriction.

Most reported cases of hemothorax have been operated upon early in the course when loculated pockets of serum still remain and the organized peel has not yet become dense cicatrix.[19] In our case both lungs were encased in a tight, nonelastic membrane which visibly restricted *expansion* of the lungs at operation. Ventilatory function studies performed before the pericardiectomy and left pulmonary decortication, again just before the right pulmonary decortication, and a third time three months after the second operation, are presented in Table II. Had bronchospirometry been performed, it would be less difficult to distinguish the effect of pulmonary decortication from the relief of pulmonary congestion by the pericardiectomy. The marked improvement in vital capacity and maximum breathing capacity after the first operation is obvious. This patient had so much injury to ribs and intercostal muscles that his rib cage itself is the limiting factor in ventilation. This explains the failure to observe further improvement after the right pulmonary decortication. Chest films clearly show numerous rib fragments in such malposition that considerable bony bridging has occurred between the ribs, accounting for much of the thoracic immobility (Figs. 4 and 5).

*Operative Approach.*—The pericardiectomy and left pulmonary decortication in this case were performed through a posterolateral transpleural approach with the patient in the prone position (Fig. 6). This approach for pericardiectomy is preferred by us, rather than the usual anterior and lateral thoracotomies, for the following reasons:

> 1. *Anatomic:* The major portion of the left ventricle is posterior to the left phrenic nerve. With the posterior approach, all surfaces of the left ventricle—anterior, lateral and posterior—are more accessible for surgery than with the anterior approaches. In addition, fair exposure of the right ventricle and left auricle is also obtained. Since the course of the phrenic nerve is easily seen, it is less likely to be injured.

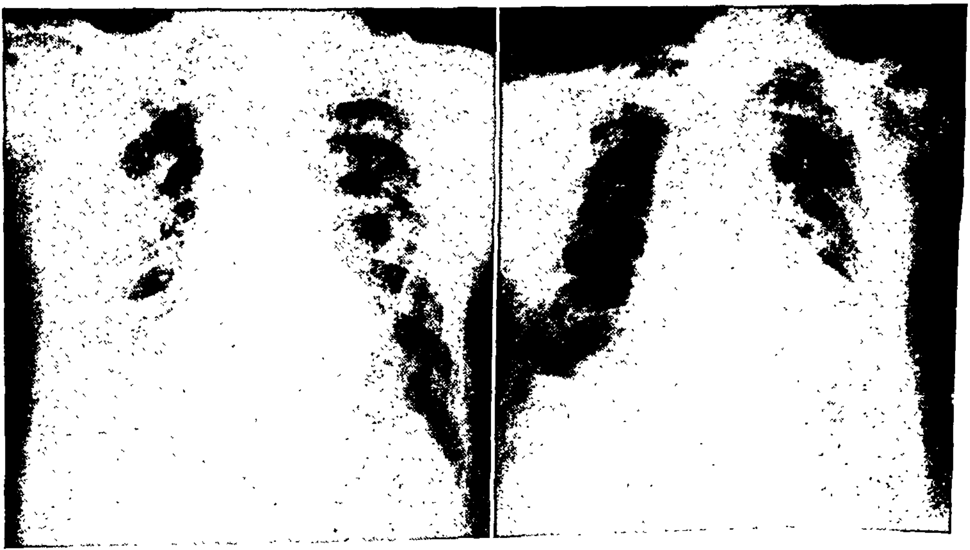

Fig. 5.—Postoperative oblique roentgenogram. There is no evidence of thickened pleural membrane.

> 2. *Physiologic:* In the lateral position, controlled positive pressure anesthesia is required to maintain adequate ventilation. The impairment of venous return, decrease in cardiac output, and lowering of the blood pressure produced by positive pressure anesthesia aggravate these same circulatory effects of constrictive pericarditis. In addition, Beecher[20] has emphasized the presence of increased amounts of respiratory acidosis in the lateral position which is not present when supine or prone is used. For these physiologic reasons, the lateral approach should not be used.

3. *Technical:* A wide exposure of operative field is obtained with the posterior approach, and the depth is about one-half that of the anterior or lateral approaches. This makes for greater ease and safety during the tedious dissection of the adherent pericardium. With the anterior or lateral position, when there is an operative accident, the hemothorax acts as a basin to collect the blood which quickly hides the defect. With the prone position, this basin is not present and immediate attention can be focused on the repair of the defect.

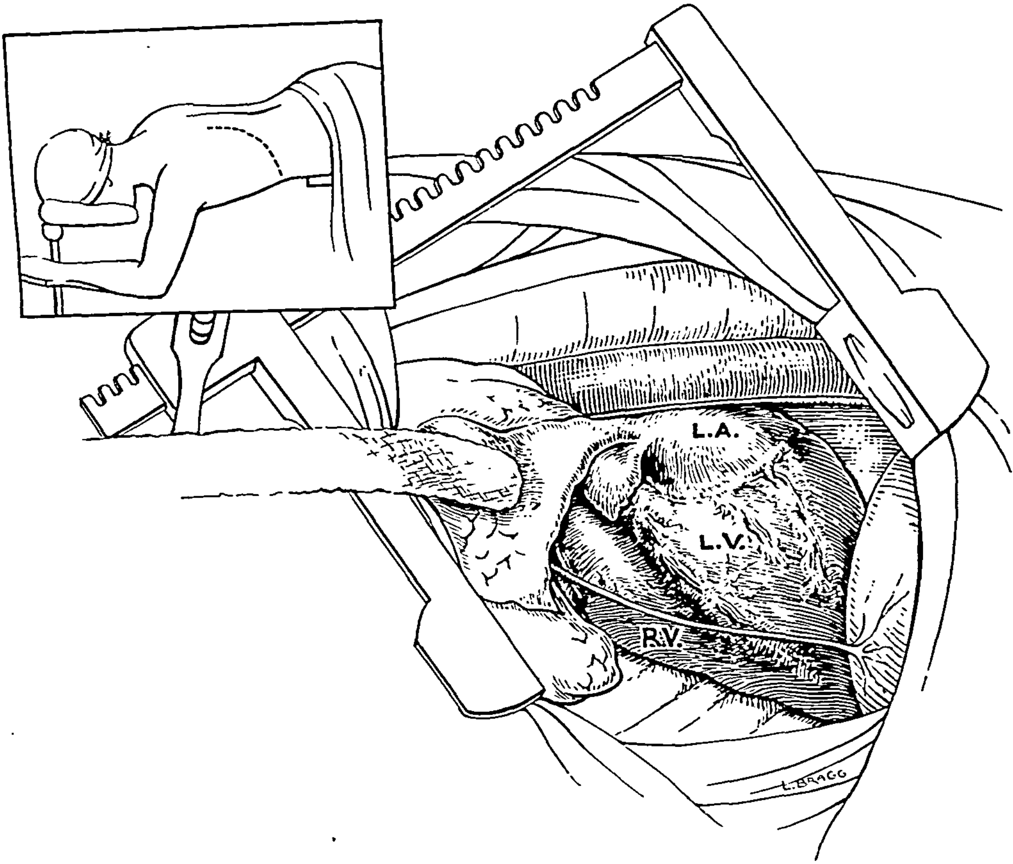

Fig. 6.—An anatomic drawing showing the excellent exposure for pericardiectomy with the patient in the prone position. The pericardium has been removed. The major portion of the left ventricle and a part of the right ventricle are posterior to the phrenic nerve. (*LA*, left auricle; *LV*, left ventricle; *RV*, right ventricle.)

*Extent of Pericardiectomy.*—In our experience, decortication of the left ventricle is necessary to insure maximum circulatory rehabilitation in constrictive pericarditis. We, therefore, practice and recommend excising the pericardium from the anterior, lateral, and *posterior* surfaces of the ventricles. The apex should be freed because it constitutes a portion of both ventricles. We have found no sound physiologic evidence to prove that the auriculo-ventricular groove should be liberated. As a general rule, no attempt need be made to free the auricles, vena cava, pulmonary artery, or pulmonary veins unless this should be indicated by catheterization studies.

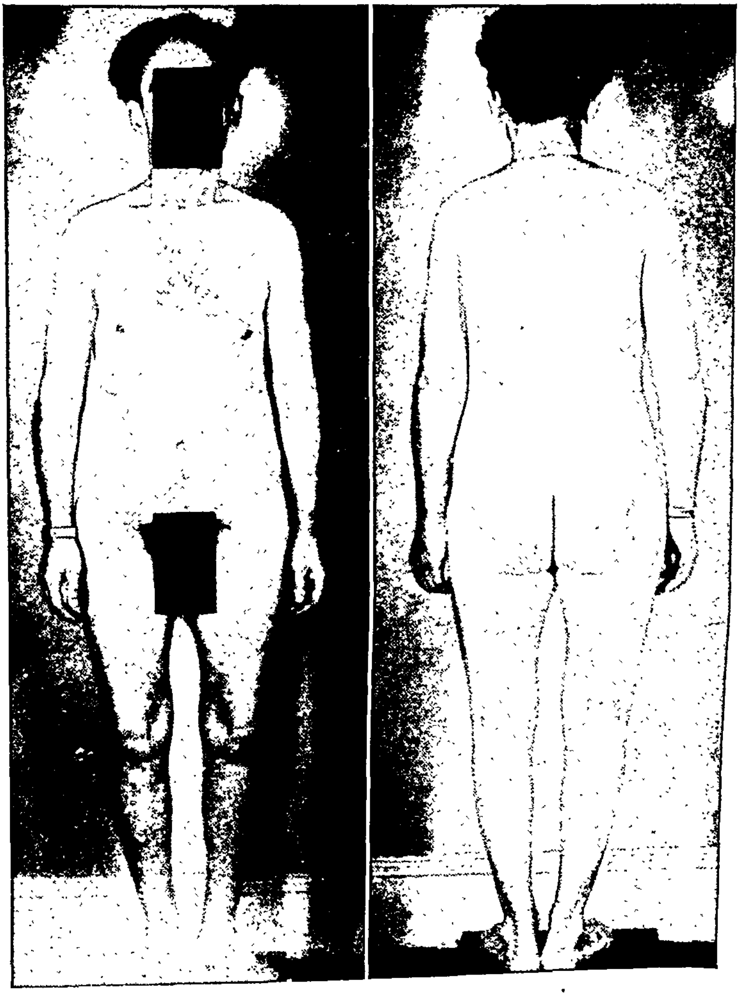

Fig. 7.—Postoperative photograph of the patient. Note the complete resolution of the edema.

## SUMMARY

1. A patient with combined constrictive pericarditis and constrictive pleuritis successfully treated by pericardiectomy and bilateral pulmonary decortication is presented. As far as can be determined, this dual constrictive disability was the result of a crushing injury to the chest with uninfected bilateral hemothorax and hemopericardium.

2. Preoperatively, cardiac catheterization clearly demonstrated impaired function of the left ventricle. Pericardiectomy was specifically directed at

total decortication of the left ventricle. Postoperative catheterization data justified this technique by showing excellent return of pulmonary vascular pressures to nearly normal.

3. A new posterior operative approach for pericardiectomy which allows excellent exposure for decortication of the posterior and lateral surfaces of the heart is described. This approach was especially suited for the combined resection of constricting membranes from both the heart and left lung in this patient.

The complete study and treatment of this patient was made possible through the close cooperation of the Boston Regional Office of the Veterans Administration.

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## DISCUSSION

DR. CONRAD R. LAM, Detroit.—I should like to congratulate Dr. Overholt and his associates for the results they obtained in this case. However, I would like to state that I think they should not generalize too much with regard to the approach in a typical case of constrictive pericarditis. I should like to bear testimony for the good results obtained by Dr. Holman's approach by median sternotomy. One can decorticate down to the phrenic nerve on each side quite easily, and the results in our hands in a small series of cases have been uniformly good.

DR. J. F. HIGGINSON, Portland, Ore.—I have had occasion to use three approaches to this problem, the circular pectoral flap approach, the left anterolateral approach which has also been described by the Boston group, and the sternal splitting approach. In contradistinction to the remarks made about how far one can reach posteriorly, it is possible by means of the sternal splitting approach to reach the back of the heart quite adequately. I think of all three approaches I have tried (I have not used this prone position) the sternal splitting approach is by far the best.

DR. EMILE HOLMAN, San Francisco.—I think this is an unusual course of events that produced this left ventricular compression, and is not comparable with the usual case of cardiac compression that we meet. I should like to state what we have found experimentally. We have had no difficulty whatsoever in producing all the symptoms of cardiac compression by constricting the right heart alone. We have four dogs for varying periods of time up to a year. Since constricting the left heart alone, we have had no symptoms whatsoever of the usual cardiac compression. I think we can prove this eventually.

DR. OVERHOLT (closing).—It was Professor Schmieden, as early as 1926, who pointed out very clearly that it was necessary to free completely the left ventricle. Since that time, there has been a great deal of argument as to whether or not the other portions of the heart should be freed or liberated. In reading over the discussion of Dr. Holman's paper which was given two years ago at the New Orleans meeting, it was apparent that the majority of discussers favored a complete exposure of the anterior surface of the heart in order to make sure that the decortication was extended to include the vena cava. In this connection, however, the observations of Dexter and Burwell are interesting. Dr. Dexter catheterized for Dr. Sweet in 1947 a patient who had been operated upon on two occasions in which the anterior approach had been used. The patient was not adequately relieved. It was demonstrated that this patient still had left ventricular constriction. Since that time, Dr. Dexter has had an opportunity to catheterize other patients in which the difficulty was found to be entirely left ventricular constriction. Dr. Sweet asked me this morning, since he could not be here, if I would not report that in his experience he has never seen any evidence that there was obstruction on the right border of the heart in cases observed at the Massachusetts General Hospital.

This particular patient that Dr. Walker has presented was unique in that the co-existing bilateral constricting pleuritis required liberation of both lung and heart from their constricting membranes. It forced us to open widely both pleural cavities and to plan the first procedure in such a way that there be complete access to the entire lung and left ventricle. The second operation afforded an opportunity to verify Dr. Burwell's and Dr. Dexter's prediction that we would not find obstruction on the right side. After decortication of the right lung, we found that there was no superior or inferior vena caval obstruction and no further decortication of the heart was required. Interestingly enough, this was our most seriously crippled patient and yet, by the posterior approach with attention to the left ventricle, our best result was obtained. Recently, we have found it necessary to reoperate upon a patient because there was an overlooked associated constrictive pleuritis, and the left ventricle had been inadequately liberated through an anterior approach. These experiences have caused us to pay more attention to the possibility of co-existing constriction of the lung as well as the heart, particularly in tuberculous subjects. It has also convinced us that the posterolateral approach in the face-down position provides a most satisfactory approach to both the left lung and posterior lateral aspect of the heart.

DR. WOODBURY (closing).—The technique of cardiac catheterization applied to patients with constrictive pericarditis has elucidated the pathologic physiology of this disease in a definitive manner which deductions from anatomy, observations at operation, and experiments in dogs have not done.

Our laboratory has catheterized eight patients who subsequently at operation were proved to have constrictive pericarditis. In none of these patients was there an increased pressure gradient between peripheral vein and right atrium. This rules out significant vena caval constriction as the cause of the elevated peripheral venous pressure. In none of these patients was there an increased gradient between right atrial pressure and right ventricular end-diastolic pressure. This rules out significant atrio-ventricular groove constriction as the cause of the elevated peripheral venous and right atrial pressures. Dr. Hansen of Copenhagen has catheterized ten patients with constrictive pericarditis, and he reports in a personal communication that he has never found pressure gradients in the great vessels indicating localized constriction at any point.

In view of this evidence against isolated stenosis, the alternative explanation for the elevated venous pressures and the diminished cardiac output in constrictive pericarditis must be embarrassment of ventricular function. That this interference is not confined to the right ventricle alone is indicated by the observation that pulmonary capillary and pulmonary arterial pressures were elevated in every one of these eighteen cases. In the eight cases catheterized in our laboratory pulmonary capillary pressures ranged from 16 to 28 mm. of mercury, two to three times the normal, and in six of the eight it was higher than right atrial pressure. Such high left ventricular filling pressures indicate that the reduced stroke volume found in every case must be due either to left ventricular failure or to limitation of left ventricular filling by a constricting pericardium.

On the basis of this evidence we are convinced that the left ventricle should be completely liberated whenever pericardiectomy is performed for constrictive pericarditis.

# THE DIRECT INTRACARDIAC RELIEF OF PULMONARY STENOSIS IN THE TETRALOGY OF FALLOT

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ALTHOUGH a considerable volume of anatomic, pathologic, physiologic, and clinical data regarding the nature of cyanotic heart diseases existed prior to 1945, these conditions aroused little interest or enthusiasm when considered therapeutically. The variety of malformations described were of so complicated a nature, the victims so pitifully afflicted, and the underlying physiologic changes so little understood or appreciated that the entire subject was relegated to a hopeless, incurable, museum-piece status. When one felt constrained to investigate more deeply, the fact that of all cardiac lesions congenital heart disease comprised but 2 to 3 per cent,[1] and of these cyanosis was present in but a relatively small number, the prospect of material contribution either clinical or technical seemed gloomy indeed. The attitude prevailed—"what is to be gained?" or "is it worth it in the final analysis?"

Such reasoning and concepts were almost immediately dispelled by the brilliant contributions of Blalock and Taussig in 1945, when the problems of cyanotic heart disease were reduced to a logical and salvagable sequence. Their initial endeavors were concerned with the mechanism whereby cyanosis was produced. They pointed out that in the common form of congenital cyanotic heart disease—the tetralogy of Fallot, embracing as it does the combination of pulmonary stenosis, dextroposition of the aorta, interventricular septal defect and right ventricular hypertrophy—the diminution of pulmonary blood flow was the dominant factor in the early demise of these infants. They had thereby reduced an anatomic malformation to its physiologic corollary. If, as it seemed at that time, it was impractical to correct the structural defects responsible for the reduction of pulmonary blood flow, it might well be feasible to counteract the result of these defects by the artificial establishment of new vascular pathways whereby unoxygenated blood might be rerouted through the pulmonary bed.

The ingenious, systemic-pulmonary shunt employing a subclavian artery-pulmonary artery anastomosis as devised by Blalock and Taussig[2] has become the standard operative method for the relief of the tetralogy of Fallot syndrome,

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and its acceptance is universal. The equally ingenious modification of this procedure, the direct aortic-pulmonary artery anastomosis of Potts, Smith, and Gibson[3] has shared the acclaim afforded such shunts, and is also widely employed by surgeons in this country and abroad. As is well known, such extracardiac shunts essentially by-pass the intracardiac pulmonary obstruction, redirecting mixed venous-arterial aortic blood into the pulmonary vascular bed where its oxygenation effectively relieves the clinical signs and symptoms manifested by the now highly publicized "blue baby." Thus an additional (fifth) defect has been added to four already present, albeit one which in large measure overcomes the immediate and obvious effects of the condition under consideration. Nevertheless, a "patent ductus arteriosus" has been created, a defect which were it to be found in existence alone would demand, by present standards, early surgical correction. The late effects of such an arteriovenous fistula on the heart and circulation are well known, and, although the interventricular septal defect in the tetralogy may conceivably counteract in some measure the deleterious late effects of an artificially produced ductus, there is certainly cause for real concern as to the eventual fate of a patient harboring such a nest of dangerous anomalies.

Without detracting in the least from the excellent results achieved by extracardiac shunt procedures, it would seem entirely reasonable to redirect one's thinking from purely physiologic lines and to consider the problems posed by the underlying anatomic defects themselves. Is it possible to relieve the pulmonary obstruction by a more direct intracardiac approach and thereby reduce (and not increase) the number of defects with which the patient must contend? In order to effectively accomplish such an obviously desirable feat it is, of course, essential to obtain a thorough understanding of the nature of the pathologic obstruction in the pulmonary channel. Having ascertained and assessed the pathologic changes, means for rapid, safe, and efficient correction must be perfected. Initial results of such a new departure as would be embodied by a direct surgical approach must be at least comparable to those already obtained by shunt procedures, and the prospect that the patient will have a more nearly permanent relief and a more certain future must at least theoretically outweigh present doubt which exists concerning the eventual physical havoc that may result from the presence of a surgically-produced shunt.

In spite of the fact that a very large number of cyanotic patients with various types of pulmonary obstruction have been studied clinically and treated surgically, there is little detailed data available as to the relative incidence of the individual anatomic variances which may be present within the right ventricular outflow tract or at the root of the pulmonary artery. It is well established, however, that pulmonary stenosis, either valvular or infundibular, may exist as a single defect or more commonly be found in association with any combination of intracardiac anomalies, particularly those designated by the term tetralogy of Fallot. In this condition, infundibular stenosis, i.e., a muscular hypertrophy or fibrous malformation of the ventricular myocardium proximal to the pulmonary valve, rather than valvular, has been considered to be almost invariably present. There is no doubt that some less common forms of infundibular stenosis almost defy direct surgical attack. On the other hand, others invite it, and certainly valvular stenosis is well suited to direct incisional separation. The crux of the situation simply stated would seem to be—how often is the stenosis valvular and how often is it infundibular? What forms does infundibular stenosis take? What is the nature of valvular stenosis? Can both be overcome surgically in a satisfactory manner with sufficient regularity to warrant the use of a direct approach routinely?

Brock[4] has devoted considerable energy to the study of the pulmonary artery-conus segment in cyanotic heart disease in the hope that clarification might lead to a direct and more logical approach. His findings and subsequent conclusions are brilliant examples of the very basic role played by exacting pathologic observation in the development of any new surgical procedure. He has described the following variations as depicted very diagrammatically in Fig. 1: (*a*), pure valvular stenosis, (*b*), a high degree of pulmonary artery hypoplasia with a bicuspid valve and stenosis lying immediately subvalvular in a narrowed muscular channel, (*c*), a high infundibular stenosis proximal to a relatively normal pulmonary artery and valve (the commonest type), and (*d*), a low infundibular stenosis at varying levels, creating a small or large infundibular chamber (third ventricle) proximal to a normal pulmonary artery and valve. The wall of this chamber may be either thin and fibrous or muscular in nature.

These observations, some obtained by direct examination of specimens and others gleaned from the existing literature, portray the over-all picture in generalities only but give no specific statistical data as to their incidence in a given number of cases. It is to be noted that of Brock's twenty-six cases of tetralogy of Fallot treated by the direct cardiac approach up to mid 1950,[5] fifteen were associated with valvular pulmonary stenosis, and eleven had an infundibular block to the pulmonary flow. There were three deaths in each group, an outstanding achievement at this early stage in the development of a new and revolutionary operation. He has stated that infundibular stenosis is more common than valvular stenosis in such cases, hence the incidence as recorded in these twenty-six cases cannot be taken at face value, for they were not consecutive cases.

A review of the early descriptions of Sandefort,[6] Corvisart,[7] Bartholemim,[8] Abernethy,[9] Farre,[10] Peacock,[11] Fallot,[12] and Keith[13] has shed no further light on the subject. Hope[14] described one case of tetralogy in an 8-year-old child whose pulmonary artery orifice was contracted "to the size of a goose quill," which is suggestive of a valvular stenosis. In Fallot's original papers[12] the first case showed a flattened and lengthened *infundibulum* forming a narrowed channel. The pulmonary artery was conical in shape with the base of the cone at its origin. The pulmonary cusps were fused and contained a small orifice. Keith[13] examined more than two hundred seventy cases of malformed hearts. One hundred forty-one of these were "imperfect because of a lesion connected with the transformation of the bulbus cordis (infundibulum) during the development of the embryo.'' At least forty-seven of these, and probably a number more, had some degree of valvular stenosis. His material is not sufficiently detailed to allow a more accurate and exacting breakdown.

Selzer and Carnes[15] analyzed a group of twenty-eight autopsied patients who had survived more than fifteen years. Valvular stenosis was present in eight, hypoplastic pulmonary artery with two pulmonic cusps in seven cases, subvalvular stenosis was present in eight cases, and separate-conus-chamber type in one. The pulmonary artery was described as dilated in five cases, and was normal or hypoplastic in thirteen.

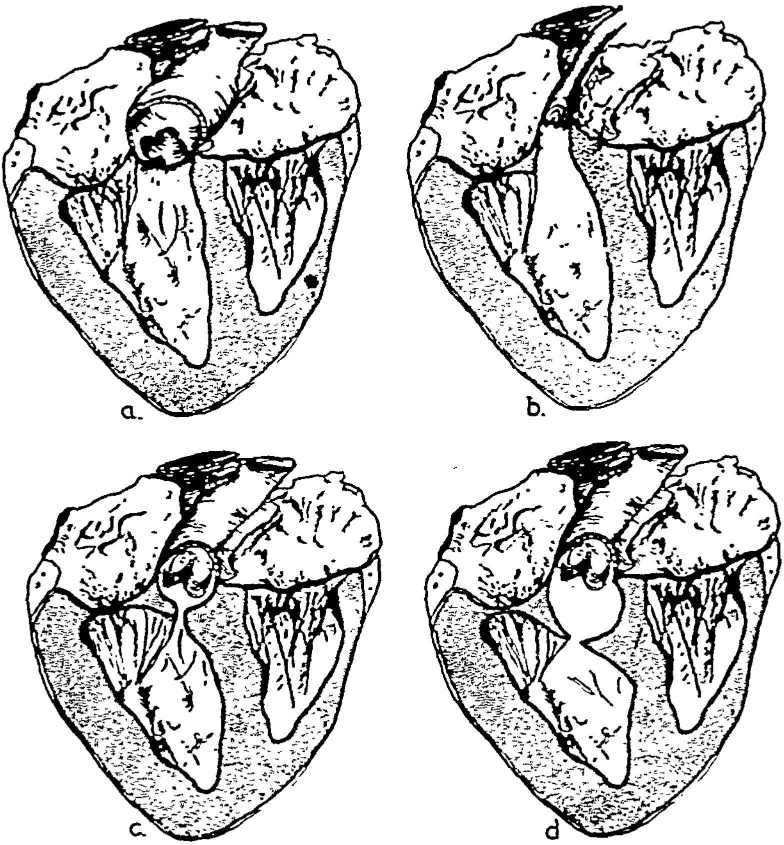


Fig. 1.—Types of pulmonary obstruction encountered in the tetralogy of Fallot: *a*, pure valvular stenosis; *b*, pulmonary artery hypoplasia with bicuspid valve and subvalvular stenosis; *c*, high infundibular stenosis with normal valve; and *d*, low infundibular stenosis with normal valve. The other pathologic components of the tetralogy of Fallot are not shown (*b*, *c*, and *d*). (Reproduced by permission of J.A.M.A.)

Taussig,[16] Blalock and Kiefer,[17] Potts, Gibson, Riker, and Leininger,[18] Parker,[19] Edwards,[20] and others feel that in the majority of instances the pulmonary stenosis in the tetralogy of Fallot is in the pulmonary conus of the right ventricle, the infundibulum, rather than in the valve itself.

Doyen,[21] in 1913, was the first to attempt the direct relief of the pulmonary stenosis in the tetralogy of Fallot. His case had a subvalvular stenosis, and did not survive the procedure. Sellors[22] was the first to revive the direct cardiac approach, and performed the first successful pulmonary valvulotomy on record. His patient was also suffering from bilateral pulmonary tuberculosis, and at last report was progressing satisfactorily in a sanatorium.

A complete review of the literature has not been included in this report, but will be the subject of a future communication.

At the onset of the present study herein reported, the authors were fully cognizant of the existing pathologic concepts and the prevailing opinion concerning these data. Nevertheless, it was considered feasible and altogether desirable to determine at first hand the practicability of employing direct intracardiac measures regardless of current impressions, no doubt prodded by a measure of success obtained in the surgical treatment of mitral stenosis acquired in the face of overwhelming odds based on time-honored pathologic and technical misconceptions.[23] This attitude was stimulated not a little by the recent visit of Brock to this country, and by his initial writings. The authors agreed, and it is still their opinion, that the pulmonary stenosis under consideration by embryologic design is *primarily infundibular* (development of bulbus cordis inclusive of the conus arteriosus). It is essential to remember, however, that the main pulmonary artery, the aorta and their contained valves, share in this developmental process. Every conceivable variation can occur, and indeed has been reported, so that malformations can exist in the muscular infundibulum, and/or the valves and/or the pulmonary artery singly or in combination. Perhaps in the majority of instances a measure of stenosis is present in the right ventricular outflow tract, but this narrowing is one of *degree* and may or may not play a part in the production of the clinical picture. The channel may be long and narrow throughout, or it may be constricted at *either end*. The channel may be quite adequate, although narrowed by normal standards, for the passage of blood with the obstructing factor localized in the valvular deformity or in the pulmonary artery itself. Only by observation and examination of the heart in the *living state* can these changes be fully appreciated from the functional standpoint. In the past, too many dictums have been proclaimed as the result of post-mortem examination which may be deceptive or represent extreme states, especially when death occurred at an early age. It must be realized that the heart as a whole and in part is a muscular walled tube undergoing marked variation in size with systole and diastole. Even though anatomically imperfect, only a part of this imperfection may be the underlying causative factor. Considerable experience with and direct scrutiny of the malformed area will be required before the exact pattern—anatomic and functional—can be properly evaluated and most effectively corrected.

## TECHNICAL CONSIDERATIONS

The technique employed in this series is a modification of that originally described by Brock and by Sellors.

The patient is placed in the supine position and anesthetized. A left anterior submammary incision is used. This permits rhythmic inflation of both lungs without embarrassment to the operator in his intracardiac manipulations.

Great care must be taken with hemostasis, since the blood of these cyanotic individuals does not readily clot. In two of the cases it has been necessary to reopen the thorax to stop persistent bleeding from the edges of the pericardial incision and from the chest wall. The extensive vascularity of all tissues, particularly in older individuals with cyanotic heart disease, greatly aggravate this tendency to bleeding.

The third intercostal space is widely opened, the costal cartilages being fractured or cut, if necessary. The internal mammary vessels are divided between clamps and ligated. The left lung usually becomes slightly collapsed and remains out of the operator's field, even when partially inflated by positive pressure.

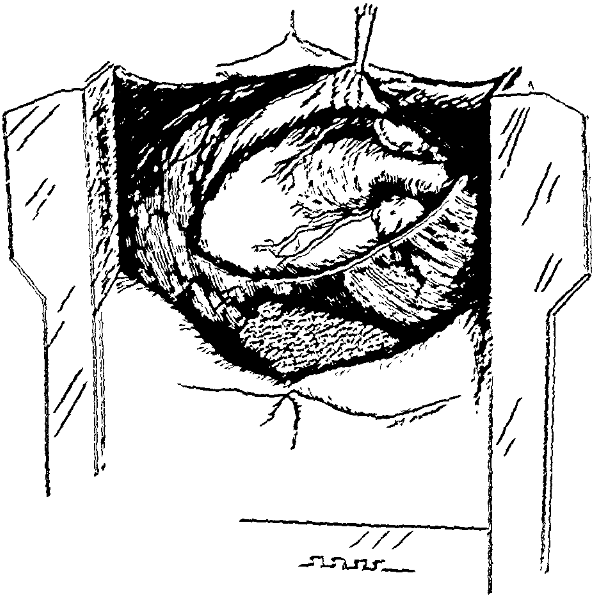


Fig. 2.—Fusiform dilatation of pulmonary artery with moderate constriction at its origin from right ventricle. Suggestive of valvular stenosis.

Care must be taken that the lower blade of the fixed rib spreader does not compress or traumatize the cardiac apex. The pericardium is picked up by a fine hemostat, one centimeter anterior to the left phrenic nerve, and incised. The incision is made parallel to the nerve, and extends superiorly over the pulmonary artery. It is advisable to limit the lower extent of this pericardial opening so as to preserve its protective action against overdistention of the ventricles, and to prevent a tendency to displacement of the heart out of the sac. However, one must have enough room to perform the essential maneuvers within the heart, and this may require extension of the pericardial incision to the diaphragm.

Several cotton or silk sutures are placed in the medial lip of the pericardial incision, and are attached to the rib spreader in such fashion that the sac is held open and the heart is somewhat rotated from its midline position toward the left. This rotation brings more of the right ventricle into view, and the exposure may be increased by converting the original longitudinal opening into a L-shaped one by adding a medial extension at the lower extremity.

The field is now inspected and palpated in an attempt to make an exact anatomic diagnosis. A fusiform dilatation of the pulmonary artery with definite constriction at its origin from the right ventricle is almost pathognomonic of a true valvular stenosis (Fig. 2). Palpation of such a dilated vessel will reveal general flaccidity, or lack of tension, within it, except at the moment of cardiac systole. A systolic thrill is palpable over the vessel. In the young subjects with dilated pulmonary arteries, there has been noted a localized systolic bulge in the vessel wall, and at this point the thrill is most intense. In older patients this bulging is absent or minimal. Digital compression of the vessel from without, at this point, sometimes permits palpation of a small volcano-like structure at the cardiac end of the vessel. This erupts at each systole with a jet of blood from the heart. More vigorous pressure against the "crater" of this "volcano" completely prevents the entrance of blood into the artery and removes all trace of the thrill (Fig. 3). Because of the high interventricular septal defect, the right ventricle still has an adequate outlet and, therefore, is not immediately embarrassed either by this digital obstruction or by subsequent complete obstruction from within by dilating sounds. Sometimes, even in a well-developed pulmonary valvular stenosis, the artery is not enlarged, and occasionally may even be unusually small. In such a case there may be a high infundibular stenosis in the immediate subvalvular region. Such a high infundibular stenosis, alone or combined with a valvular stenosis, is probably not too uncommon, and

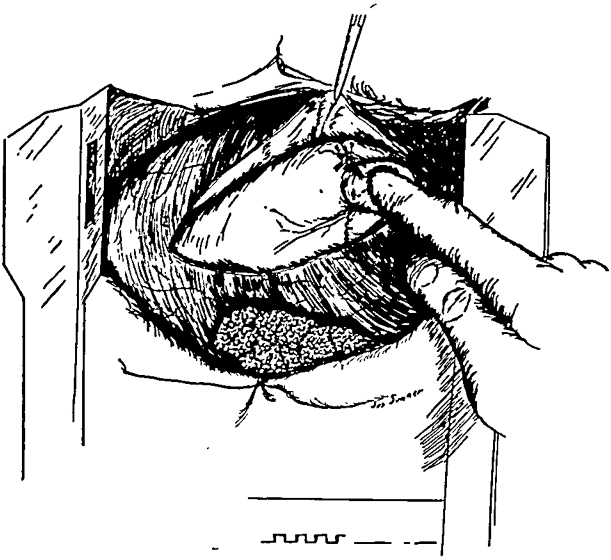


Fig. 3.—Digital compression of the pulmonary artery to determine the presence of valvular stenosis.

*may have been present in a number of cases which we interpreted as being valvular.* Such a defect would respond well to the usual treatment applied to the true valvular type, and so does not seriously complicate the surgical management.

In certain instances, fortunately rare in children more than 1 year of age, intrapericardial inspection will reveal a generally hypoplastic main pulmonary artery, or one with a stricture or coarctation at some point along its course. The thrill will then be felt along the course of the vessel distal only to the point of obstruction.

In cases of infundibular stenosis, which may make up approximately half of the cases seen after 1 year of age, the pulmonary artery is usually small. It may, however, fan out or funnel out slightly at its distal extremity. The thrill will be felt rather diffusely along the course, but will also usually be felt over the distal ventricle. This thrill over the ventricle tends to differentiate the condition from cases of valvular or arterial stenosis which do not present a thrill over the ventricle.

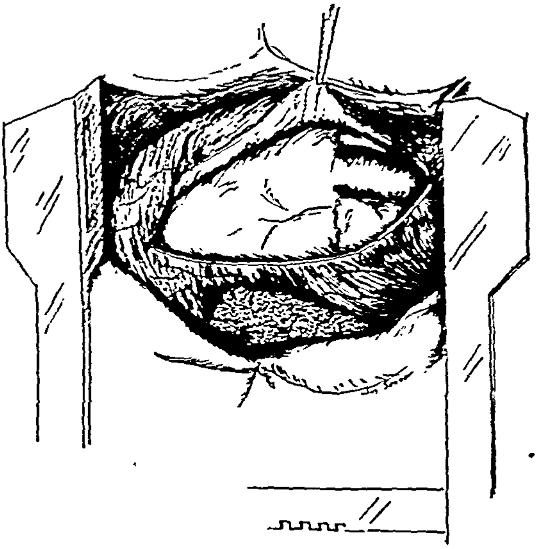


Fig. 4.—Variable sized bulge outlining the underlying infundibular chamber. Small vascular channels frequently overlie the segment of true constriction.

Sometimes a thin-walled infundibular chamber can be made out distal to an "isthmus" or waistlike narrowing of the right ventricle (Fig. 4). This may be a very small chamber varying from a pea to a cherry in size. Or it may be a sizeable structure, the size of an egg. In other cases it is just as thick and muscular as the true right ventricle. In such cases the diagnosis is less easily made by inspection. Even here, however, there may be a waistlike effect. Visible blood vessels running in a direction different from the course of the normal coronaries may outline this "waist" and its underlying septum (Fig. 4).

In no case should an opening be made into a thin-walled infundibular chamber, as the tissues may not be strong enough to hold reparative sutures. A muscular infundibular chamber may, however, be entered with impunity.

When a valvular stenosis is clearly present, or strongly suspected, we inject the anterior wall of the right ventricle with 0.5 per cent procaine solution to lessen the likelihood of producing a ventricular arrhythmia. Two transverse stay sutures of heavy silk are then inserted deeply into the myocardial wall so that it can be incised in a *transverse* direction about 2.5 cm. below the level of the valve. A very tiny transverse opening is then made, and a small urethral sound is inserted through it until it enters the ventricular chamber. Extreme care must be taken that this sound does not pass far beyond the endocardium, lest it pass below some of the papillary musculature attached in this region. It is then directed up toward the valve and through it into the pulmonary artery. In a small child a No. 18 or 20 standard sound will usually become tightly gripped by the stenotic valvular orifice (Fig. 5). Having passed the sound through the valve, the direction for passing the valvulotome is established, since it can be bent in a curve similar to the sound. The sound is now withdrawn until its tip is close to the endocardial wall, and is redirected so that it may be passed down toward the apex of the right ventricle. Failure to meet intracardial obstruction rules out the presence of any significant infundibular septum. It is then passed up into the aorta if the diagnosis of tetralogy has not previously been established. If the operator is now satisfied as to the absence of an infundibular and the presence of a valvular stenosis, the sound is removed from the heart, and bleeding from the puncture wound is controlled by digital pres-

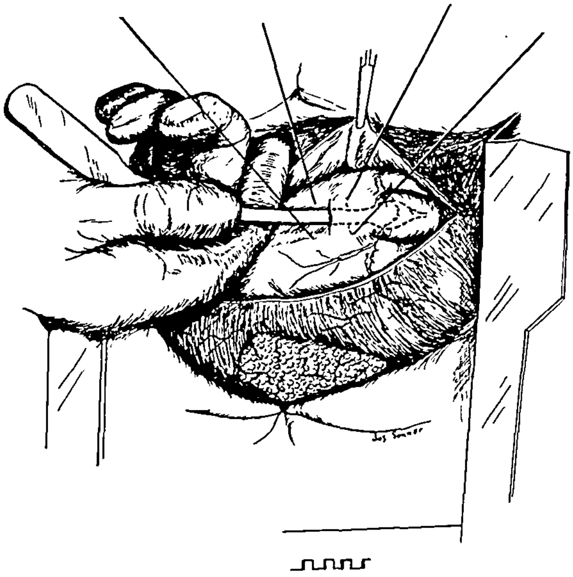


*Fig. 5.—Malleable probe, or urethral sound, passed into and through the tiny valvular orifice.*

sure and traction on the stay sutures. If there remains any doubt as to the exact anatomic diagnosis, the myocardial incision is enlarged, and a finger of the left hand is inserted into the right ventricle. The little finger is chosen in children, but the index finger is more adept and, therefore, preferable in adults. It can be inserted through the pulmonary valve if not stenosed, through the high ventricular septal defect, and back to the tricuspid valve region of the ventricle. This method of digital intracardiac examination has much to commend it, and little but psychologic inhibitions to prevent its much wider application.

Having established the presence of a valvular stenosis, the operator will choose a suitable sized valvulotome of the Brock type. We prefer the so-called "universal valvulotome" (G. P. Pilling & Sons) because the flat malleable handle permits adjustment to various anatomic variations and causes less bleeding from the heart (Fig. 6). It is our practice to estimate the proper sized valvulotome with relation to size of the pulmonary artery at the level of the constricting ring. We then select the next smaller instrument. Failure to choose a smaller valvulotome than the exact estimated size once resulted in division of the entire thickness of the arterial wall as well as the valve ring.

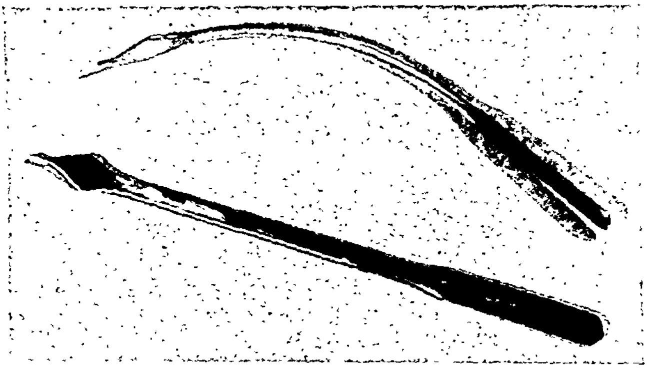


Fig. 6.—Flat malleable universal valvulotomes used for division of valvular stenosis. These can be molded to fit the conditions present in individual cases.

While such an inadvertent incision of the lateral or medial walls of the pulmonary artery may be readily sutured, unnecessary blood loss and additional risk to the patient is the natural result. The possibility of such an accident should moreover point the advisability of incising the valve in a horizontal plane. If the valvular division should be made in a vertical plane, the possibility of inadvertent injury to the posterior (inaccessible) wall of the artery, technically nearly impossible to suture, would be ever present.

Having chosen the valvulotome, it is inserted through the myocardial incision. Brock usually passes his knife so that the anterior myocardial opening is longitudinal, rotating the instrument ninety degrees after entering the cardiac chamber. We prefer to have the instrument enter the right ventricular chamber through a transverse myocardial incision since it then, by slight upward redirection as soon as it enters the chamber, is already in position to enter and cut the pulmonary valve without danger of dividing a papillary muscle. The valvulotome is smoothly passed until the olivary tip seeks out and passes through the tiny apical valvular orifice. By further passage the somewhat cartilaginous resistance of the valve is overcome, and the instrument passes freely up into the lumen of the pulmonary artery where it becomes readily palpable through the anterior arterial wall (Fig. 7).

The instrument is then removed from the heart, and bleeding is controlled by digital pressure, perhaps with slight upward countertraction with the stay sutures. A few minutes should be permitted to elapse to observe the response to this maneuver. Then, we feel that *one* dilating instrument should be passed through the pulmonary valve to obtain the widest possible aperture without further rupture of the valve or vessel. Brock has a special dilator which he recommends. An ordinary urethral sound of adequate size is most satisfactory in our hands. Probably the left little finger or the left index finger is the best of all dilating instruments at this stage, permitting tactile orientation and appreciation, as well as manual enlargement of the opening (Fig. 8).

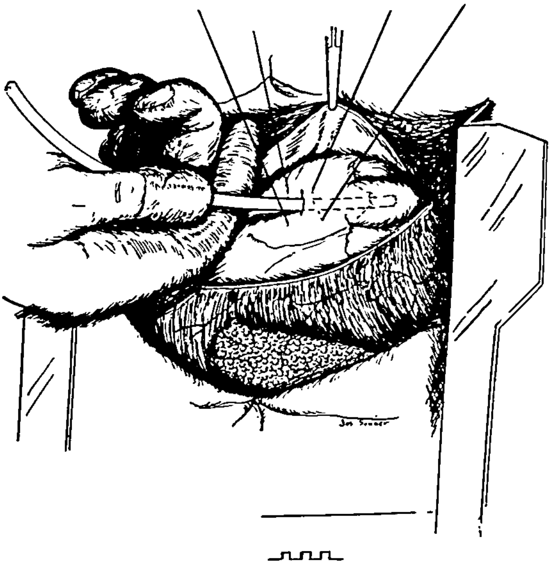


Fig. 7.—The valvulotome dividing the stenosed valvular cone into a biscupid valve.

After dilatation, the finger or instrument is removed from the heart, the stay sutures are crossed to control bleeding, and interrupted cross sutures are applied to approximate the cut muscular edges. The stay sutures may be tied or removed. The pericardium is reconstructed at least to cover the line of myocardial incision. Very careful hemostasis is employed in closing the chest because of the previously mentioned tendency to bleeding. The pleura is always drained by a No. 26 urethral catheter connected with a water-seal bottle.

The technique for the relief of an infundibular obstruction is, of course, significantly different from the foregoing but may be as satisfactory in suitable circumstances.

Having inspected and palpated the exposed heart, the operator should have a very definite opinion as to the probable existence of an infundibular stenosis, and an idea of the probable location of the septum. Stay sutures are applied into the wall of the myocardium as previously described to permit a *longitudinal* incision. Brock chose to enter the heart either well above or below the infundibular septum, so that his punch can be passed through the diminutive natural communication of the two chambers, to punch out a circular collar of tissue from the septum. We chose, on the other hand, to make our incision directly over the infundibular septum so that we can bite or rongeur out a sizeable portion of it. Naturally, we take great pains not to extend any of the myocardial incision over a thin walled infundibular chamber which is sometimes present.

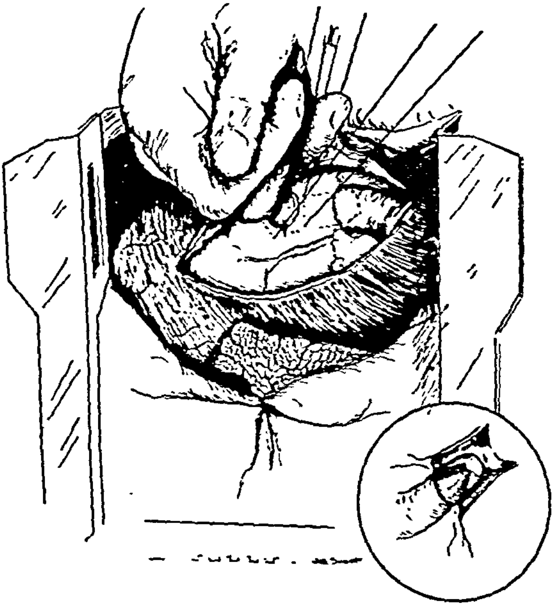


Fig. 8.—The left index finger, or largest urethral sound, passed through the divided valve to insure patency, not to produce additional dilatation or splitting.

Usually a small knife puncture is made at the selected point, and a small urethral sound is inserted through it into the heart. By passing it successively up through the pulmonary valve, through the high septal defect into the aorta, and again down toward the apex of the ventricle, an idea of the nature of the obstruction can be obtained. Better yet, the left little finger, or even the index finger, may be inserted into the heart and passed successively in the same directions. Usually, one of two situations is found. There is either a high subvalvular membranous stenosis, or there is a transverse muscular septum at a lower level, and usually it is immediately under the middle of the myocardial incision if that has been correctly placed.

In the former instance (high stenosis), while the finger is in place, the proper sized flat-handled valvulotome may be passed alongside the finger and up through the obstruction out into the pulmonary artery in a manner similar to that used for valvular stenosis, but this time guided by the intracardiac finger. After dividing the stenosis, the instrument is removed and the finger is passed up into the pulmonary artery as a dilator.

In the lower infundibular stenosis the exploring intracardiac sound or, preferably, finger reveals the transverse muscular septum. If the myocardial incision is directly over it, it may be extended to better straddle the septum. A pair of modified bone rongeurs (Fig. 9) is then plunged into the heart, jaws open to engage the septum. A good-sized bite of tissue is thus removed, usually widely interrupting the continuity of the obstructing septal wall (Fig. 10).

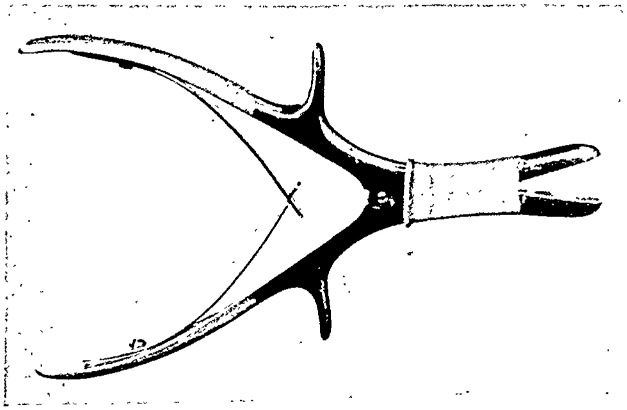


Fig. 9.—Small double-action rongeur with sharp cutting edges and deeply umbilicated to hold the resected specimen.

The use of this ronguer is most suitable in older children or adults. In infants and small children we prefer a small-sized pituitary forceps as suggested by one of us (D. F. D.). The operator's finger controls bleeding from the heart just before and after the rongeurs are inserted. It may be considered necessary to remove a second bite of septal tissue in the same way. The finger now momentarily re-enters the heart, to reveal complete continuity of the infundibular and true right ventricular chamber. The stay sutures are crossed and the myocardial incision is closed as previously described in the valvular case.

In the case with extreme hypoplasia or atresia of the pulmonary artery, no direct relief of the obstruction will be possible. Such cases must always be treated by either a Blalock-Taussig or a Potts-Gibson systemic artery-pulmonary artery anastomosis. However, there are some cases in which the pulmonary artery at the level of the valve seems to be of normal size. About a centimeter above the valve the vessel becomes abruptly narrower like an aortic coarctation, only to again become normally large at the level of its bifurcation. The thrill in such cases is felt in the artery distal to the stricture. This condition may be treated by passing successively larger sounds through the stenosed area until it is felt that blood flow has been substantially increased. The same type of myocardial incision is used as has been outlined.

## ANALYSIS OF CASES

An analysis of thirty consecutive cases of tetralogy of Fallot treated surgically by the direct intracardiac approach forms the basis of this report. Not included are our cases of "pure" pulmonary stenosis, usually valvular, in which there is an intact interventricular septum without overriding of the aorta. A separate report of this series will be presented in the near future.

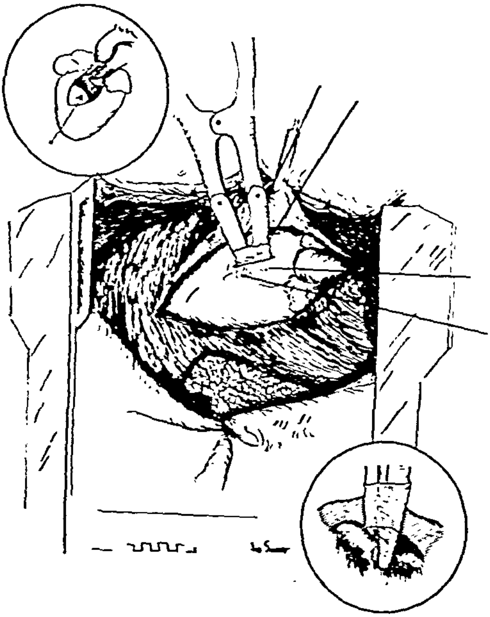


Fig. 10.—The rongeur in place showing method of resection of a portion of the infundibular ridge.

As a group, these patients exhibited all of the classical clinical features of the often-described tetralogy of Fallot. Easy fatigue, cyanosis of the lips and nail beds, exaggerated by physical activity, clubbing, and dyspnea on the slightest exertion, were characteristic. In addition to the usual clinical findings, electrocardiographic pattern, fluoroscopic and roentgen picture, the diagnosis in twenty-four of the thirty was confirmed preoperatively by the angiographic demonstration of immediate and simultaneous opacification of the aorta and right ventricle. In four of the remaining six (Cases 8, 16, 20, 22, 23, 28) the study was attempted but proved unsatisfactory. The diagnosis in these cases was verified at the time of surgery by direct observation or by the passage of a probe through the ventricular septal defect into the aorta from the right ventricle. The first eight cases have been published in abstract elsewhere.[24]

TABLE I. CASES OF TETRALOGY OF FALLOT TREATED BY DIRECT INTRACARDIAC SURGERY

| CASE | AGE AND SEX | TYPE OPERATION | DATE OPERATION | TYPE STENOSIS | AORTIC ARCH | HBG.—R.B.C. (PREOP.) | HBG.—R.B.C. (POSTOP.—OVER 6 MO.) | RESULTS TO DATE |
|---|---|---|---|---|---|---|---|---|
| 1. M. B. | 3—F | valvulotomy | 12-7-49 | valvular | L | 19.0 8.7 | 15.0 5.1 | excellent |
| 2. K. K. | 3—M | dilatation | 2-3-50 | infundibular | L | 16.7 5.6 | 13.9 4.9 | very good |
| 3. D. J. | 4—F | resection | 2-7-50 | infundibular | R | 16.4 5.6 | 11.8 4.5 | excellent |
| 4. H. C. | 12—M | valvulotomy | 3-10-50 | valvular | L | 16.2 5.5 | 11.1 3.9 | excellent |
| 5. M. R. | 18—F | valvulotomy | 3-29-50 | valvular | L | 15.0 4.7 | 12.0 3.8 | died 2½ mo. (transfusion reaction) |
| 6. L. F. | 3—M | resection | 4-12-50 | infundibular | L | 16.0 5.3 | 12.5 4.2 | very good |
| 7. R. B. | 6—M | valvulotomy | 4-26-50 | valvular | R | 14.5 4.3 | 11.5 3.9 | very good |
| 8. A. S. | 21—F | resection | 5-3-50 | infundibular | R | 18.0 7.4 | 14.0 4.8 | excellent |
| 9. W. B. | 2—M | resection | 7-24-50 | infundibular | R | 12.2 5.4 | 11.7 4.0 | excellent |
| 10. T. P. | 16—F | valvulotomy | 8-23-50 | valvular | L | 18.6 6.6 | | very good |
| 11. C. P. | 2—F | dilatation | 8-28-50 | pul. art. sten. | L | 11.8 6.6 | 13.0 4.4 | excellent |
| 12. M. O. | 2—F | attempted resection | 9-26-50 | infundibular | L | 15.9 7.0 | | died on table (trauma and hemorrhage) |
| 13. E. U. | 4—M | valvulotomy | 9-28-50 | valvular | L | 15.2 5.3 | 10.7 5.6 | excellent |
| 14. R. D. | 5—M | dilatation | 10-11-50 | pul. art. sten. | L | 16.0 6.1 | 13.0 3.8 | excellent |
| 15. B. H. | 4—F | attempted resection | 10-23-50 | infundibular | R | 16.9 5.8 | | died on table (cardiac arrest) |
| 16. D. B. | 21—M | valvulotomy | 10-25-50 | valvular | L | 10.0+ 5.3 | 76% 4.3 | very good |
| 17. E. K. | 12—F | valvulotomy | 10-28-50 | valvular | R | 17.7 5.5 | | excellent |
| 18. S. H. | 2—F | valvulotomy | 11-3-50 | valvular | L | 18.6 5.7 | | died on table (cardiac arrest) |
| 19. W. B. | 2 mo.—M | valvulotomy | 11-8-50 | valvular | L | 17.0 6.7 | | died—6 hr. (postop.?) |
| 20. R. H. | 15—M | dilatation | 11-15-50 | pul. art. sten. | L | 18.0 8.8 | | excellent |
| 21. W. G. | 2—M | attempted resection | 11-20-50 | infundibular | L | 14.9 4.9 | | died on table (cardiac arrest) |
| 22. K. S. | 2—F | resection | 11-23-50 | infundibular | L | 17.7 7.4 | | very good |
| 23. S. B. | 36—M | dilatation | 11-29-50 | valvular | L | 18.0 7.0 | | very good |
| 24. J. G. | 22—M | valvulotomy | 11-22-50 | valvular | L | 18.6 6.9 | | very good |
| 25. W. C. | 31—M | attempted dilatation | 2-28-51 | infundibular | L | 16.9 6.1 | | died on table (tracheal obst.—anesthesia) |
| 26. R. M. | 15—M | valvulotomy | 3-14-51 | valvular | R | 16.5 5.9 | | excellent |
| 27. J. W. | 21—M | valvulotomy | 3-14-51 | valvular | L | 16.0 6.3 | | excellent |
| 28. L. K. | 12—F | valvulotomy | 3-28-51 | valvular | R | 15.0 5.5 | | too recent for evaluation |
| 29. M. F. | 14—F | valvulotomy | 3-28-51 | infundibular (subvalvular) | L | 83% 4.3 | | too recent for evaluation |
| 30. D. S. | 4—M | resection | 4-9-51 | infundibular | L | 11.1 4.8 | | too recent for evaluation |

Protocols of the more recent cases are similar in type except for the usual individual variations and will not be enlarged upon here. Table I contains the essential data in each instance.

### VALVULAR STENOSIS

Surprisingly, in fifteen (50 per cent) of the group, ready egress of blood flow from the right ventricle was obstructed by *stenosis of the pulmonary valve,* a much higher incidence than has been presumed to occur in this combination of defects.

The presence of a valvular stenosis could not be predicted preoperatively with certainty. Their preoperative clinical course did not vary appreciably from the group as a whole. The position or character of the usual systolic murmur over the precordium was not diagnostic. Laboratory studies shed no light as to the type of pulmonary block. Catheterization studies done in a few yielded high pressures (50 to 80 mm. Hg) in the right ventricle and low pressures in the pulmonary artery (5 to 30 mm. Hg) when entered. The site of obstruction could not be accurately determined by observing the passage of the catheter in its course through the right ventricle fluoroscopically.

Angiocardiographic examination was perhaps of greater value than all other studies combined. As a rule, although not invariably true, the presence of valvular stenosis could be strongly suspected by the demonstration of a large pulmonary artery in which the diodrast tended to "hang up" throughout the entire study on successive films (six to eight seconds). Post stenotic dilatation of the pulmonary artery was observed but not with the frequency and clear delineation frequently seen in "pure" pulmonary stenosis with intact ventricular septum.

Direct inspection and palpation of the heart and great vessels through the open pericardium at the time of surgery provided the most definitive answer as would be expected although in several instances even this examination left some doubt as to the exact nature and location of the obstructing deformity (see above, under technique).

The average age at the time of surgery in this group was 13 years, the oldest patient being 36 years, and the youngest 2 months. Nine were males and six females. There was a normal left aortic arch in eleven, right aortic arch in four.

The results obtained could not have been more dramatically divergent. These patients either did beautifully or did not survive. Six were obviously *excellent,* and this was apparent within a short time after surgery. Their present activities are *unlimited* and, except for a faint, scarcely noticeable (to the suspicious observer) bluish tinge to the mucous membranes of the mouth after vigorous exercise, they have no cyanosis. Five are regarded as *very good.* These cannot quite carry on complete normal activity in all its phases for their individual age group. As the lapse of time from the date of surgery in the oldest of these is slightly under a year, there is every reason to believe that further improvement can be expected. One additional case (Case 28) is progressing exceptionally well at this time, but an insufficient period has elapsed to warrant classification of the result.

There were two operative deaths. In the first (Case 18), a child of 2 years, the pulmonary artery was found to be very small. Cardiac action deteriorated as the pericardium was opened. It was feared that the insertion of a Brock knife into this small pulmonary artery could not be accomplished with safety. Accordingly, a dilating sound was inserted through the stenosed valve. Cardiac action ceased and, in spite of vigorous resuscitative measures, the child died.

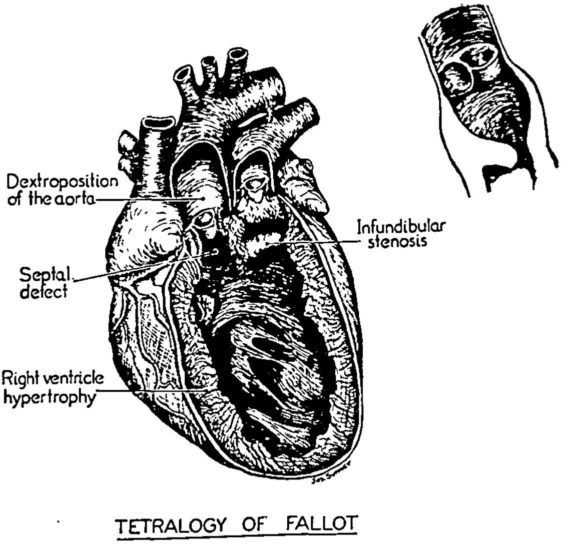


Fig. 11.—High infundibular stenosis with grape-sized infundibular chamber proximal to a normal valve. (Drawn from specimen.)

The second patient (Case 19) was a 2-month-old infant with hydrocephalus. The pulmonary artery was somewhat smaller than normal. A valvulotomy was performed, and the infant's color became pink almost immediately. The entire procedure from skin to skin required only twenty-five minutes. Only 10 c.c. of blood were lost. About one hour postoperatively he exhibited some respiration distress. He was bronchoscoped, and a small amount of mucus was aspirated. Thereafter, his condition was eminently satisfactory for five hours. Then, suddenly, over the course of ten minutes, he deteriorated. Cardiac action was strong, the lungs were clear, and there was no obstruction to the airway which could be demonstrated bronchoscopically. Nevertheless, his respirations became weaker and weaker, and finally ceased. Autopsy was limited to the heart. It was found that the stenosed pulmonary valve had been converted into a bicuspid valve, and an adequate lumen existed. It is hypothesized that, in view of the hydrocephalus, the infant died of central causes.

A third patient (Case 5) died thirteen weeks postoperatively. Her demise was directly attributable to a blood transfusion reaction following a second operation, but it must be attributed ultimately to the initial pulmonary val-

valvulotomy. Following incision of the valve, too large a sound was passed into the pulmonary artery. Pulmonary and tricuspid insufficiency became severe, and several weeks postoperatively plication of the pulmonary artery in the region of the valve was accomplished in an effort to decrease the lumen. A severe transfusion reaction occurred postoperatively and, after a stormy course of ten days, the patient died.

## INFUNDIBULAR STENOSIS

An obstructing malformation of the pulmonary conus area (infundibulum) was present in twelve instances. For the most part the distortion of the right ventricular outflow tract followed a set pattern, although it varied sufficiently to permit classification into four categories based primarily on the size of the infundibular chamber and the position of the obstructing tissue.

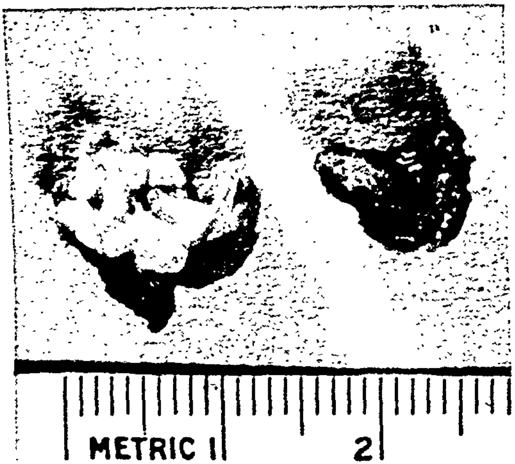


Fig. 12.—Type of specimen removed by rongeur resection. To the left, a portion of muscular ridge and overlying endocardium at the infundibular orifice. To the right an additional section of muscular septum removed to insure complete patency. (Photographed from specimen.)

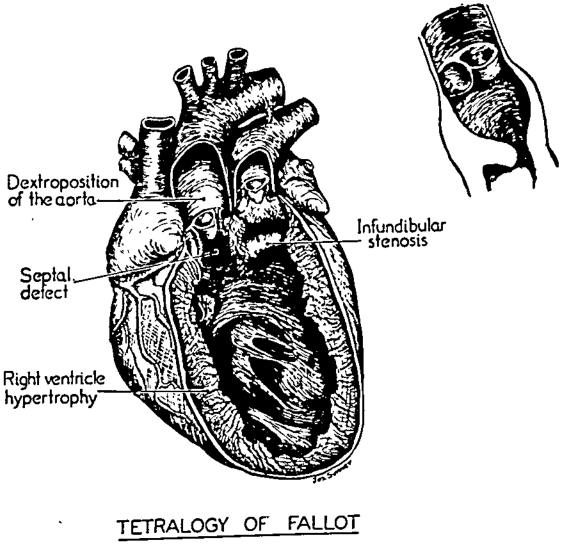
1. Six cases (50 per cent) in this group were of the common type designated in Brock's classification above (Fig. 11). These had a high infundibular stenosis with a grape-sized infundibular chamber proximal to a normal valve. (Cases 2, 3, 6, 8, 15, 21). The point of muscular and fibrous obstruction was narrow, and lent itself readily to resection (Fig. 12). Two of the four patients in the infundibular series who died had this type of obstruction, and are commented upon below.

2. Three cases were subvalvular in type, and were so immediately proximal to the valve that differentiation from true valvular pathology was difficult (Cases 9, 22, 29). Two were resected and one was opened with the valvulot after the manner used in valvular stenosis (Fig. 13). There were no d

3. In two cases there was a high degree of pulmonary artery hyp with a long, narrow muscular channel proximal to a split-pea sized subv

further improvement can be expected. One additional case (Case 28) is progressing exceptionally well at this time, but an insufficient period has elapsed to warrant classification of the result.

There were two operative deaths. In the first (Case 18), a child of 2 years, the pulmonary artery was found to be very small. Cardiac action deteriorated as the pericardium was opened. It was feared that the insertion of a Brock knife into this small pulmonary artery could not be accomplished with safety. Accordingly, a dilating sound was inserted through the stenosed valve. Cardiac action ceased and, in spite of vigorous resuscitative measures, the child died.



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There were four operative deaths. In the first (Case 12), a girl of 2 years, the heart slowed and stopped as the pericardium was opened. After five minutes of rhythmic manual contractions fortified by a small intracardiac injection of adrenalin and atropine, a vigorous heartbeat was restored. The pulmonary artery was quite small, the branch to the left lung being greatly atrophic. The usual myocardial incision was made, one bite taken from the muscular obstruction with difficult and more than average manipulation and blood loss. Heart contractions ceased as the myocardial incision was being sutured. Efforts toward resuscitation were ineffective. Post-mortem examination of the heart revealed an infundibular muscular channel with a very tiny chamber proximal to a normal valve (Fig. 14). In all probability this type will be most difficult to relieve directly by methods now in use. A shunt procedure could not have been performed on the left in this case.

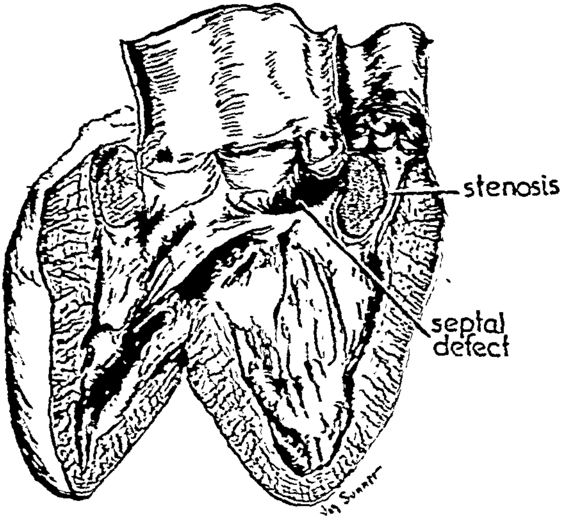

Fig. 14.—High degree of pulmonary hypoplasia with a long narrow muscular channel proximal to a split-pea sized subvalvular chamber. (Drawn from specimen.)

Case 15, aged 4 years, was a girl who had multiple and severe congenital anomalies in addition to her cardiac malformations. She was deaf, mentally deficient, and had peculiar bony deformation of her rib cage. The pulmonary artery was soft, flabby, and of normal size. A jet could be felt on the anterior surface of the pulmonary artery, even though the obstruction was infundibular. A bite was attempted by rongeur, but little was removed. A second bite seemed more satisfactory, and the myocardium was sutured. The heart slowed, but of the infundibular patient is the poorer, and that fewer live to an age where the best surgical result can be obtained because of their more complicated malformations. This thought is not borne out, however, by analysis of Brock's series, although his cases were selected rather than consecutive. Six were males, and an equal number females. There was a normal left aortic arch in eight, right aortic arch in four.

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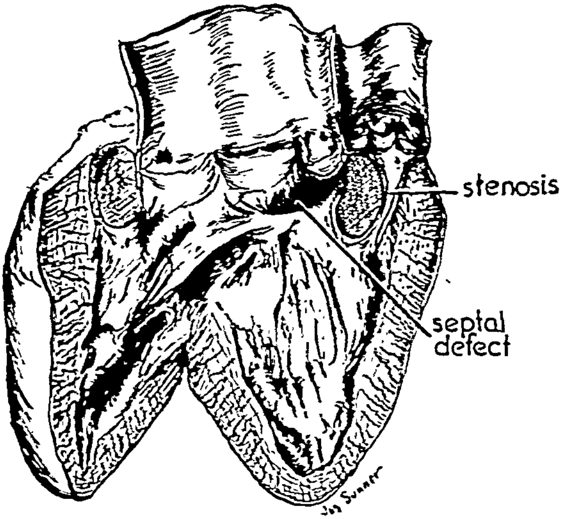

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The third patient (Case 21), a boy aged 2 years, was in a very precarious condition, and the operation was performed as a semi-emergency. This patient was deaf, dumb, and blind, with microphthalmia and microcephalia. The pulmonary artery was small, and a slight thrill could be felt on its anterior surface and over the distal portion of the pulmonary conus. The myocardium was opened directly over the obstructing infundibular ridge, an adequate bite was removed in the right place, and the wound closed (Fig. 16). The procedure was carried out with dispatch with only average trauma and blood loss. Within a minute the heart stopped and could not be revived. It is possible that the cerebral changes in this patient could not tolerate this or any other type of surgery. Examination revealed that a proper bite had been taken. There was an adequately functioning bicuspid valve.

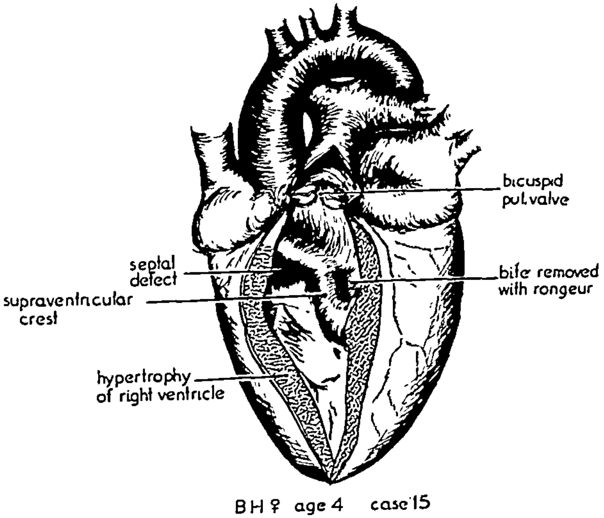

Fig. 15.—A resection of infundibular taken erroneously proximal to supraventricular crest. (Drawn from specimen.)

The last patient (Case 25) was a boy, aged 4 years, in reasonably good condition. The pulmonary artery was small, and a soft thrill could be felt over it and the distal ventricular outflow tract. The nature of the infundibular obstruction was confusing. The myocardial incision was made over the narrowed muscular channel, and a finger inserted to dilate the long narrow channel. The opening was felt to give and partially split in a *stellate manner*. The incision was closed, the heart slowed. Gentle massage quickly restored a normal beat, and the chest was partially closed. Difficulty in maintaining adequate respiratory exchange was experienced, and shortly thereafter the heart stopped and could not be revived. A complete plug was found blocking the intratracheal tube. Examination of the specimen revealed that the dilatation had been off center, and a split had been made in the ventricular septum (Fig. 17).

### STENOSIS OF THE PULMONARY ARTERY

There were three cases in which a localized constriction occurred in the main pulmonary artery distal to a normal pulmonary valve but proximal to the

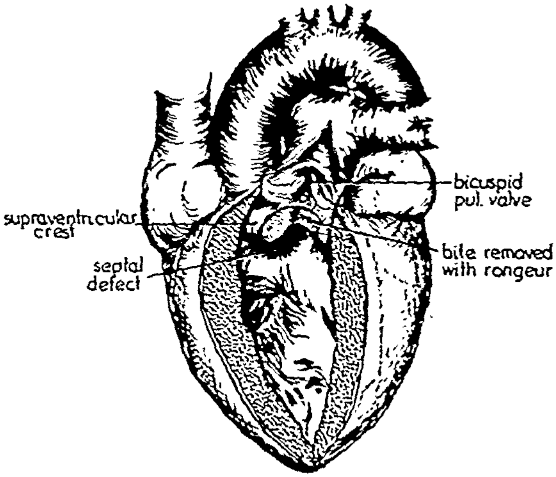

*Fig. 16.*—An adequate resection of a portion of the infundibular ridgelike obstruction. (Drawn from specimen.)

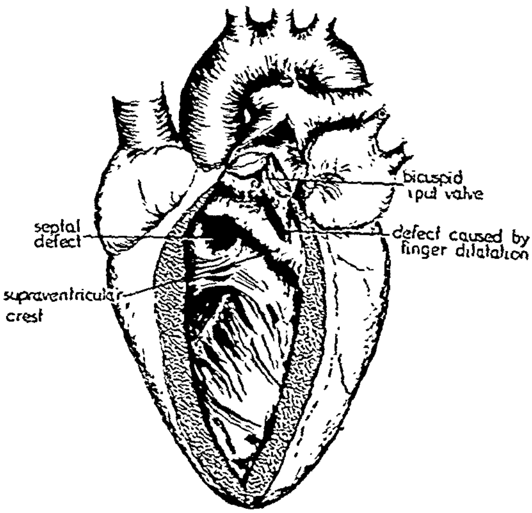

Fig. 17.—Digital dilatation splitting in infundibular tissue in the wrong area leaving the supraventricular crest intact. (Drawn from specimen.)

bifurcation of this vessel into its right and left major branches (Cases 11, 14, 20) (Fig. 18). These patients presented the identical clinical picture of the other patients in this series, nor were there any points of variation in their routine diagnostic studies. An angiocardiogram was successful in only two of the three in this group. The pulmonary artery appeared small in both, and no suggestion of the constriction found at surgery could be detected in it either preoperatively or at re-examination after surgery.

Direct inspection and probing of the ventricular outflow tract and pulmonary valve at the time of surgery failed to reveal any evidence of significant obstruction to the flow of blood except in the localized area as described above. The introduction of graduated sounds seemed to accomplish a most satisfactory enlargement of the arterial lumen. The eventual fate of a dilatation procedure such as this must await the verification of time.

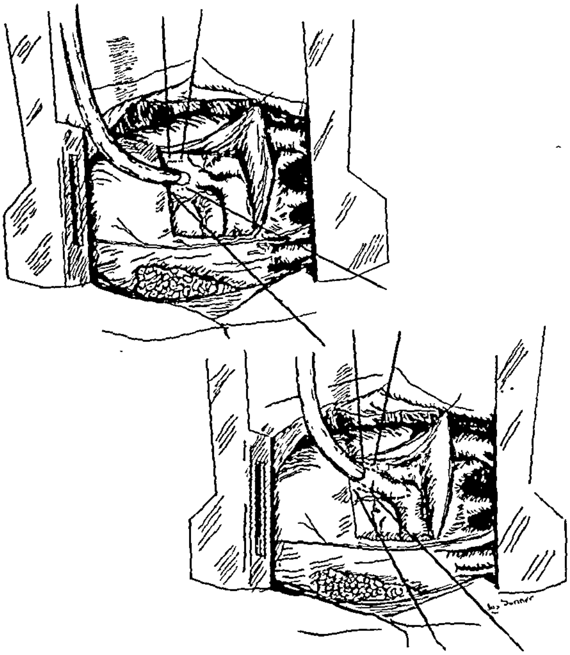

Fig. 18.—Localized constriction of the main pulmonary artery distal to a normal pulmonary valve. The method of dilatation employing graduated urethral sounds.

The average age at the time of surgery in this group was 7 years, the patients being 2, 5, and 15 years, respectively. Two were boys, one a girl. In each there was a normal left aortic arch.

## DISCUSSION

In these thirty cases there were seven deaths, a mortality rate of 23.3 per cent. One death (Case 5) occurred thirteen weeks postoperatively, as described above, so that the actual operative mortality was 20 per cent. In the valvular group the mortality was 20 per cent, but if Case 5 is not considered an operative death, the rate drops to 13.3 per cent. The mortality in the infundibular group was 33.3 per cent. There were no deaths in the three cases with localized stenosis of the pulmonary artery.

This mortality rate, although high, is quite in keeping with the early results obtained with anastomotic procedures. Blalock[25] reported an over-all mortality of 22 per cent in the first one hundred forty-four patients, with a diagnosis of pulmonic stenosis subjected to the production of systemic artery-pulmonary artery shunt. It is reasonable to assume that a material reduction in this initial rate will be brought about as further experience in the selection of cases and in the operative technique is gained; just as it has in the extracardiac procedures.

Many of the difficulties encountered in the intra- and postoperative management of these cases have been and will further be resolved as additional experience is gained. The most disconcerting intraoperative complication has been cardiac arrest. This occurred in twelve patients (Cases 2, 3, 8, 12, 15, 18, 21, 22, 23, 25, 25, 29). Eight of these recovered through the use of proper measures, and some now demonstrate the most satisfactory clinical response in the series. Of the four who did not recover, two had associated severe cerebral anomalies; it is recognized that such patients as a rule withstand any type of surgery poorly. The relatively high incidence of this serious complication is probably due to the following factors: (1) improper technique; the surgeon was hampered by his lack of knowledge as to the exact anatomical situation, or, although this had been determined, he was overeager in his manipulations, with resultant overwhelming trauma to the heart; and (2) the patients were not chosen with regard to their suitability for any operation; they were chosen because they had the tetralogy of Fallot and could be helped by operation—that was the single criterion.

One case of cardiac arrest may be cited as being of particular interest. This was the oldest patient in the group (Case 23). Exposure of the pericardium was delayed because of the necessity of clamping and ligating innumerable dilated vessels in the thoracic wall. The pericardium itself was covered by a network of dilated, tortuous vessels. As it was being opened, cardiac arrest occurred. Despite this, a myocardial incision was made. The valve was quickly dilated manually and with sounds. The myocardial incision was sutured and alternate manual compression, and release of the heart instituted. Adrenalin and atropine were administered into the ventricle. Myocardial tone improved and a spontaneous beat was established. Ventricular fibrillation ensued. Electric shock (110 volts alternating current) caused a severe tetanic convulsion and cardiac arrest. The heart then spontaneously resumed a normal rhythm. The total duration from the initial arrest to resumption of normal rhythm was nine minutes. Four and one-half hours after his return to the ward there was such brisk hemorrhage through the drainage tube that he was brought back to the operating room and his chest reopened. Innumerable bleeding points in pericardium and thoracic wall were ligated or cauterized. His subsequent course was marked by aphasia and a right foot drop. Both improved steadily, and although there still remains some verbal difficulty he may be considered to have derived remarkable benefit from surgery.

In the immediate postoperative period, the most challenging complication has been the development of signs of cardiac failure. This occurred in eight patients (Cases 2, 3, 5, 6, 7, 8, 22, 24).

Each of these individuals, with one exception (Case 2), was either allowed ambulation or assumed the privilege before one week had elapsed. While the principle of early ambulation is accepted after most surgical procedures, it is not to be recommended when a cardiac chamber has been entered via a myocardial incision. We now feel that a period of at least two weeks should lapse before the patient is allowed any but minimal activity.

Minor pleural effusions have been encountered but have not posed a special problem.

In spite of the disappointing results in some patients and the complications encountered in others, it must be concluded that the direct intracardiac approach to the problem of pulmonary stenosis, whether it be pure or a part of tetralogy of Fallot, would appear to be sound and logical from both the anatomic and physiologic standpoints. There are some cases which, because of their nature, will require a shunt, but these should be determined only after thorough intrapericardial assessment of the anatomic situation.

## SUMMARY AND CONCLUSIONS

1. Our total experience with the direct intracardiac relief of pulmonary stenosis in the tetralogy of Fallot has been presented. Thirty consecutive cases over a period of sixteen months have been analyzed.

2. Representative literature pertaining to the various types of pulmonary obstruction which may occur in the tetralogy of Fallot has been reviewed. Information concerning the relative incidence of infundibular and valvular stenosis is scant. Of thirty consecutive cases of tetralogy of Fallot, reported herein, the pulmonary stenosis in fifteen was thought to be valvular, infundibular in twelve, and localized to the pulmonary artery in three.

3. The technique of the direct intracardiac approach for each type of pulmonary obstruction has been presented, and the results assessed.

4. It has been pointed out that the currently accepted anastomotic procedures for the relief of pulmonary stenosis in the tetralogy of Fallot are attended by certain dangers because an additional vascular defect has been produced. The direct approach, if it be technically feasible, would appear to offer a more satisfactory long-term solution to the problem.

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## DISCUSSION

DR. WILLIS J. POTTS, Chicago.—Dr. Glover would be disappointed if we did not discuss this paper. In speaking of pulmonary stenosis, we must very definitely differentiate between the two distinct types; *pulmonary stenosis with intact intraventricular septum*, and pulmonary stenosis of the typical tetralogy of Fallot with septal defect. Briefly, a word about the first group:

(Slide.) Description of these instruments has not been published, and it might be interesting to spend a minute describing them. We have what we call an expanding valvulotome, made so that the wound in the heart may be kept small. We have borrowed from the urologists the principle of a retreating or sheathe blade. The dilator is built on the same principle. Indicators on the instruments show how widely the valvulotome or dilator is open.

(Slide.) Here you see both instruments completely opened. This basket type of dilator is made so that when it is in the pulmonary artery completely expanded, blood can still flow through the vessel. This is seemingly rather important.

(Slide.) The operative approach is a left submammary incision. The closed valvulotome is thrust into the right ventricle. Then by palpation and observation of the pulmonary artery we determine how widely to open the valvulotome.

(Slide.) The valvulotome is thrust through the stenotic pulmonary valve, closed, and withdrawn. A finger is put over the opening momentarily and the dilator is then inserted. We use the dilator to complete the division of the stenotic valve as we do not dare cut to the very edge of the pulmonary artery. The dilator can be rather leisurely expanded as the pulmonary artery is observed, and blood can continue to flow through the vessel while this is being done. The opening in the heart is closed with two stitches.

To throw some light on the subject of pulmonary stenosis with a septal defect, we analyzed 24 post-mortem hearts of patients who had died of tetralogy of Fallot. It was interesting to find that we had exactly the same percentages that Dr. Blalock presented yesterday. In nineteen of the twenty-four there was infundibular stenosis of varying degree or atresia. In four, there was combined infundibular and valvular stenosis. In only one was there complete stenosis of the valve itself. *In these five patients, incision, not excision,* would have been a valuable procedure.

It is appropriate that we bow to the courageous young men, because what they are doing is an extremely valuable stimulus to further study. However, in view of the high mortality associated with reaming out the stenotic segment, I still feel that we should do the shunt operation until one can more safely go inside the heart.

DR. BAILEY (closing).—We did not attempt to present a pathologic paper, although such might have been very desirable. We did not have sufficient pathologic material to present anything comprehensive. We presented a technical and clinical paper on thirty practically consecutive cases. You will note, both from Dr. Blalock's statistics yesterday and from Dr. Potts' today, that we are apparently out of line with the findings in an autopsy series of cases of tetralogy of Fallot. However, Brock, in twenty-six similar living cases, found a distribution of types of pulmonic obstruction almost exactly the same as ours. Our point would be that this is a living series, the type of patient you are liable to meet to take to the operating table, whereas many of the other cases have probably died at an early age and, because of the extremes of pathologic variation, did not live to be considered for surgery. We also feel that part of the discrepancy may be based upon the fact that anatomic infundibular septum may be quite different from functional infundibular septum, in that probably many hearts which seem superficially normal actually have a residual or partial infundibular septum, which a pathologist would so classify if he were looking for it. They do not obstruct the flow of blood through the pulmonary artery and so are not of clinical significance.

We realize, of course, that there is a great difference between pure pulmonic stenosis and the tetralogy of Fallot. There is no question in anyone's mind, I think, that the case of pure pulmonic stenosis is usually valvular and can be treated very well by the methods which Mr. Brock, Dr. Blalock, and Dr. Potts have so well presented. There probably is also no great question when true valvular stenosis occurs in tetralogy of Fallot, because obviously the direct technique is then quite comparable and the results should be excellent. The only argument, I believe, is with regard to the type of surgery for the infundibular type. We too feel that there is a certain group of severe infundibular stenoses which will never be suitable for the direct approach. Our suggestion is that, having opened the pericardium, an incision be made in the heart and a finger, the little finger or the index finger according to the size of the heart, be passed into the right ventricle, which seems to tolerate it well. Then the exact pathology can be pretty well determined and one can immediately tell whether there is any hope of doing a direct resection of the obstructing infundibular septum. On a theoretic basis, as Dr. Blalock pointed out yesterday, and Dr. Glover today, it would be better to diminish the number of defects by removing the pulmonary obstruction rather than increase the number by adding a patent ductus arteriosus. But that, of course, may not always be entirely practical.

I think Dr. Potts' instrument for cutting the pulmonary valve is superb, and we have already ordered one and hope to be able to use it some day. It is certainly superior to the instruments we have been using. I think the chief criticism of this paper is with regard to the present operative mortality and morbidity. We really lost seven patients, the last one several weeks postoperatively, because I inadvertently avulsed a pulmonary valve. But this presentation was of our first thirty cases. Without meaning any disrespect to the great men we have talked about, I believe Dr. Blalock's mortality in his first 144 cases was approximately the same as ours in this series, and so we hope to do better as we learn better.

# TREATMENT OF CORONARY ARTERY INSUFFICIENCY BY IMPLANTATION OF THE INTERNAL MAMMARY ARTERY INTO THE LEFT VENTRICULAR MYOCARDIUM

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MONTREAL, QUE.

IN THIS paper a preliminary report is given in human cases of coronary insufficiency which have been treated by implantation of the left internal mammary artery into the left ventricular myocardium. The experimental data upon which this procedure is based are briefly described.

Attempts to increase the circulation of the myocardium in cases of coronary artery insufficiency may be grouped as follows: (a) Vascularization through the surface of the heart by grafts and granulomas.[1-3] (b) Cardiac vein ligation.[4] (c) Arterialization of the coronary venous system.[5]

A different approach to the vascularization of the ventricular myocardium was first attempted by us in 1945.[6] The left internal mammary artery was employed as a source of fresh arterial blood for the ventricular myocardium. The internal mammary artery was transected and detached from its chest wall bed. It was then placed within a tunnel in the ventricular myocardium. An anastomosis developed between the implanted internal mammary artery and the left coronary circulation. The presence of the anastomosis was proved by injection studies, radiographs, plastic casts, and serial sections. (*Figs.* 1, 2, 3*A*, 3*B*, 4).

In our early experiments, approximately 60 per cent of implanted internal mammary arteries failed to form an anastomosis.[7] Experiments were conducted in an effort to discover some of the factors which were responsible for the anastomosis. It was found that an anastomosis between the implanted internal mammary artery and left coronary vessels occurred with greatest frequency when (1) A short length of artery is freed from the chest wall. (2) The internal mammary artery is placed in the inner two-thirds of the myocardium. (3) The sixth intercostal is left open when buried within the myocardium.

It is important to avoid trauma and infection which favor thrombosis. Thrombosis limits the size of the anastomosis. Small anastomoses, however, frequently develop through a recanalized thrombus.

Anastomoses occurred in from 50 to 75 per cent of the animals, dependent on the technique of implantation used. The next step was to determine the value of such an anastomosis in affording protection against coronary artery occlusion. This was gauged by a study of the mortality rate and the development of ventricular infarcts following ligation of the anterior descending branch of the left coronary artery.

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Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, New Jersey, April 16-18, 1951.

In a series of twenty-six animals divided into four groups, the average survival rate after anterior descending branch ligation was 50 per cent. In one group it was 75 per cent. Sixty-nine per cent of the animals surviving did so without infarction. The surviving animals all showed an extensive mam-

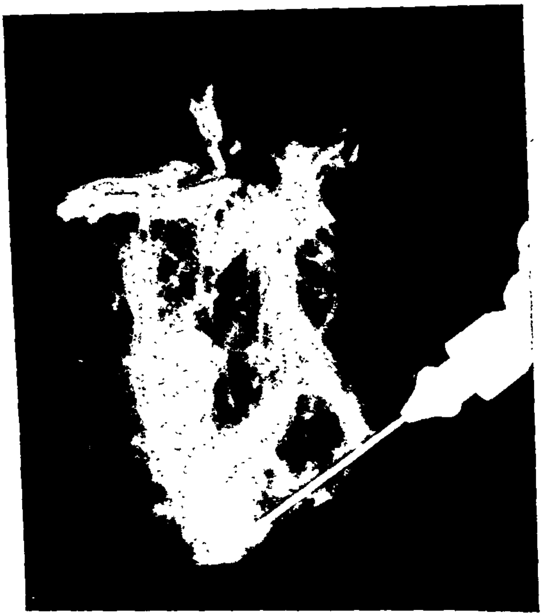


Fig. 1.—Roentgenogram of the left coronary tree, which has been filled by injecting the left internal mammary artery seventeen weeks after it was implanted into the left ventricular wall.

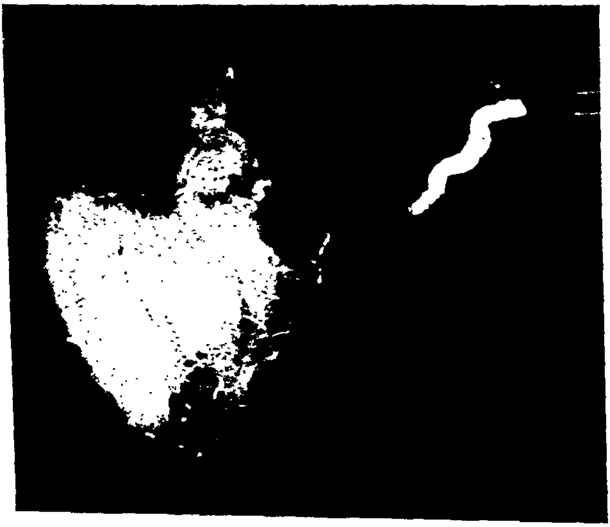
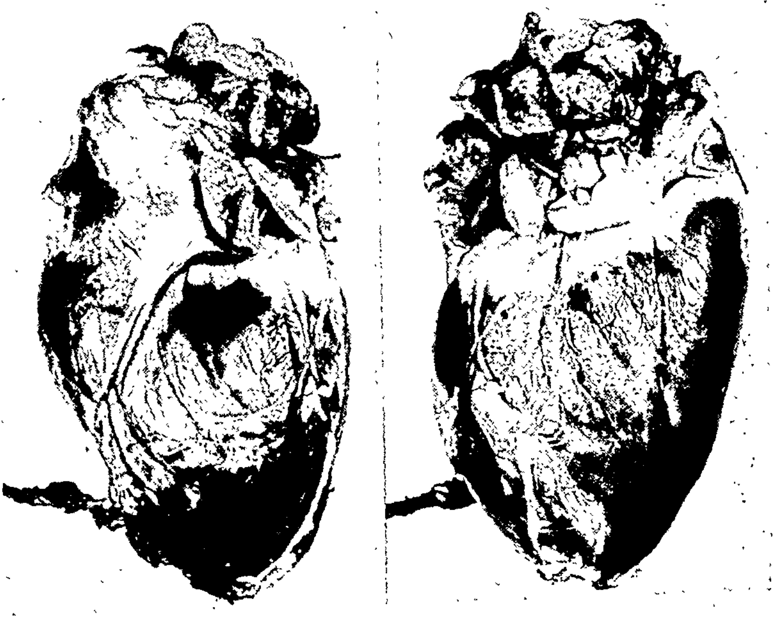


Fig. 2.—Roentgenogram shows heart of animal (162) with injection mass filling the anterior descending and circumflex branches of the left coronary artery fourteen weeks after implantation. This animal survived anterior descending branch ligation.

mary-coronary anastomosis. In one-third, the anastomosis was established through a recanalized thrombus within the internal mammary artery. In no single case has an animal died or developed infarction following anterior descending branch ligation when a large anastomosis existed.



A. B.

Figs. 3,*A* and 3,*B*.—The heart of animal (39) with blue Schlesinger's solution filling the branches of the coronary artery. The injection was introduced through a 13-week-old internal mammary artery implantation.

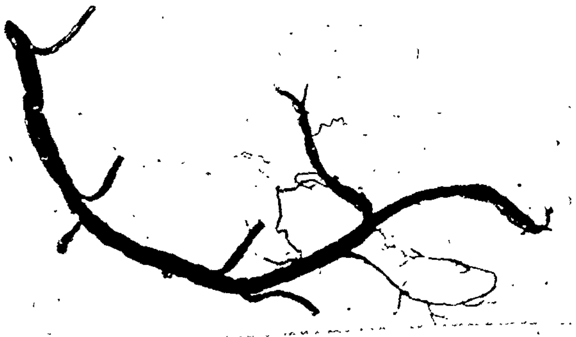


Fig. 4.—Shows a cast which has been made by injecting the internal mammary artery with a plastic substance forty-six weeks after implantation into the left ventricular wall. The small sharply cut off branches to the right are intercostals tied with cotton. The fine arborizations to the left have developed within the heart muscle.

In a control group of animals without internal mammary implantation, death occurred in 90 per cent, and a 5 by 5 cm. infarction developed in the surviving 10 per cent.[8, 9] The facts seemed to indicate that an implanted internal mammary artery is capable of carrying on the circulation of the left ventricle after coronary occlusion. The evidence, however, was largely statistical, thus further proof was sought.

### DIRECTION OF BLOOD FLOW IN INTERNAL MAMMARY ARTERY

The direction of blood flow through the implanted internal mammary artery was studied in order to determine whether it was bringing blood to the ventricular myocardium. Direct determination of blood flow in the internal mammary artery was difficult, so the indirect method was used. Animals which survived anterior descending branch ligation of the left coronary artery were subjected after four weeks to complete and sudden occlusion of the implanted internal mammary artery. If the internal mammary artery was maintaining the circulation of the left ventricle, then following its ligation, either death or infarction should result. This is exactly what happened. In three animals with an internal mammary implant which had survived anterior descending branch ligation, the internal mammary artery was ligated. One animal died within twenty-four hours, and displayed an edematous cyanotic area of the anterior wall of the left ventricle. One survived for three days before dying

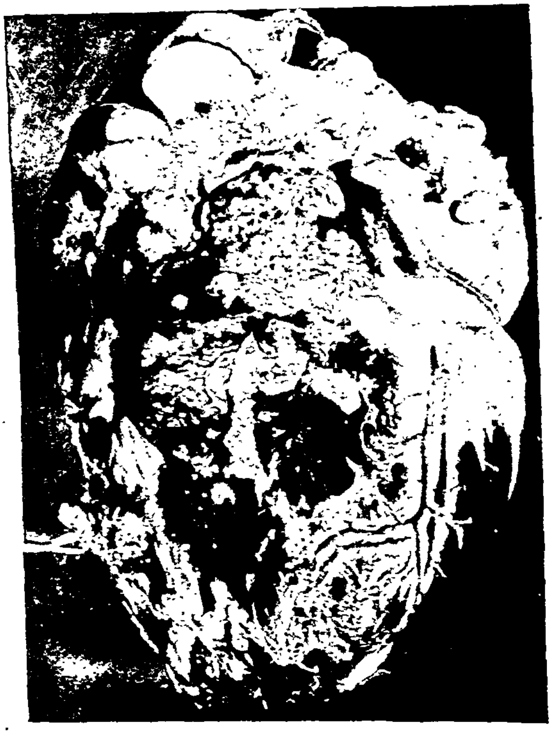


Fig. 5.—Heart of animal No. 10, M. series; this animal underwent anterior descending branch ligation sixty-seven days after internal mammary implantation and survived. The implanted internal mammary artery was ligated thirty-seven days later and the animal died of a large anterior wall left ventricular infarct.

from a large infarct in the same location (Fig. 5). The third animal survived, but examination of the sacrificed specimen revealed that multiple intercoronary anastomoses were present.

Following these experiments, there seemed little doubt as to the formation and value of a coronary mammary anastomosis. However, skepticism was expressed with regard to the character of the anastomoses and to their duration.

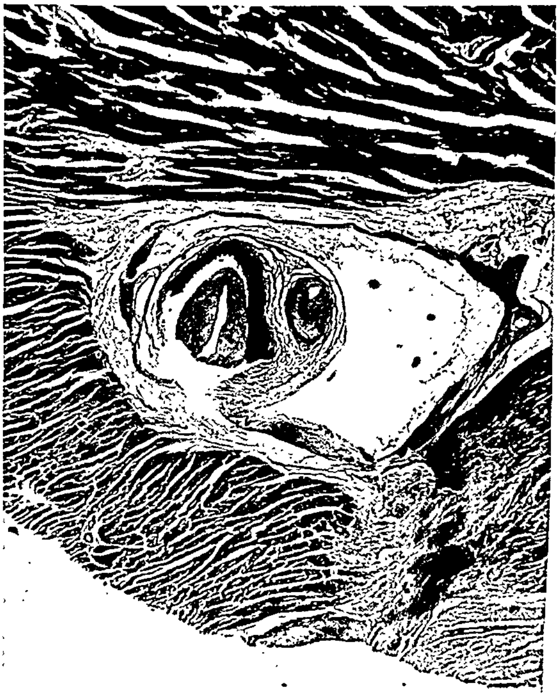


Fig. 6.—Represents a serial section of an implant lying within the muscle of the myocardium fifty-eight weeks after implantation. Note the branch lying alongside of parent vessel. This branch has been traced into the myocardium.

### DURATION OF ANASTOMOSIS

Recently, Glenn and Beal[10] confirmed the development of an anastomosis between an implanted internal mammary artery and the coronary vessels. However, they stated that the new vessels tend to disappear at the end of eight weeks. They further suggested that the new vessels were small and resembled granulation tissue.

In our series, the average interval from implant to sacrifice was eleven weeks (Table I). Some animals were kept fifty-eight weeks after implantation. The anastomoses at the end of that time not only persisted, but were large enough to protect against death and infarction following ligation of the anterior descending branch.

The anastomoses which developed have been shown by serial sections, special muscle and elastic stain studies, and by casts to be formed by true arterial branches of the implanted internal mammary artery (Figs. 6, 7, 8, 9).

The experimental results just described were obtained by implanting the internal mammary artery into normal dogs' hearts. It was suggested that in human coronary insufficiency such a procedure would be of little value because of the presence of occluded coronary vessels and associated myocardial ischemia. It was, therefore, decided to produce coronary artery insufficiency experimentally in the dog.[11] This was done by wrapping the origin of the anterior descending branch of the left coronary artery with a sclerosing type of cellophane. The

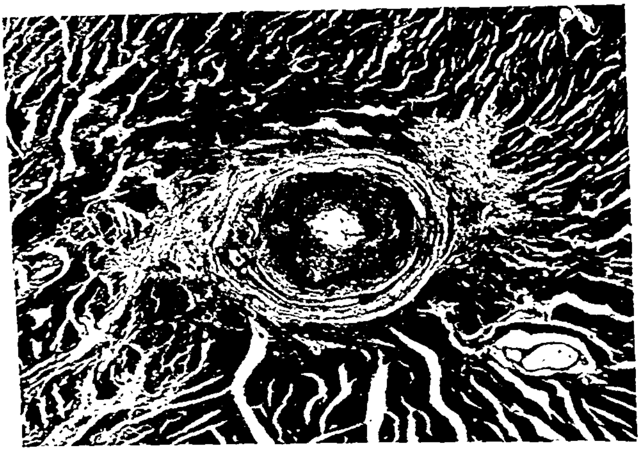


Fig. 7.—Dog 7. Internal mammary artery one hundred twenty-two days after implantation shows artery lying between heart muscle fibres with many branches. There is very little reaction around the implant and the scar tissue appears inactive. Note branch at 7 o'clock.

TABLE I. DURATION OF ANASTOMOSIS.

| TYPE OF ANASTOMOSIS | NO. OF ANIMALS | PRESENCE OF ANASTOMOSIS AFTER IMPLANT (WEEKS) | VALUE OF ANASTOMOSIS AFTER LIGATION OF ANT. DESC. BRANCH LT. CORONARY ARTERY |
|---|---|---|---|
| *Microscopic:*<br>Anastomosis small due to thickened or partially thrombosed internal mammary artery. | 1 | 5 | Protection against death or infarction afforded in 3 animals only. |
| | 1 | 6 | |
| | 1 | 7 | |
| | 2 | 8 | |
| | 2 | 11 | |
| | 2 | 12 | |
| | 2 | 13 | |
| | 1 | 14 | |
| | 12 | | |
| *Macroscopic:*<br>Anastomosis large, internal mammary artery patent. | 1 | 6 | No deaths or infarction occurred.<br>In cellophane coronary artery sclerosis marked improvement in exercise tolerance occurred. |
| | 2 | 7 | |
| | 5 | 9 | |
| | 5 | 11 | |
| | 4 | 12 | |
| | 2 | 14 | |
| | 1 | 17 | |
| | 1 | 18 | |
| | 3 | 19 | |
| | 1 | 21 | |
| | 1 | 31 | |
| | 1 | 41 | |
| | 1 | 46 | |
| | 1 | 58 | |
| | 29 | | |

cellophane surrounding the origin of the anterior descending branch of the left coronary artery set up a periarterial fibroplasia which resulted in a gradual contraction through scar tissue of the coronary artery and narrowing of its lumen. This caused a reduction in blood flow through the narrowed vessel and resulted in ischemia of the left ventricular muscle supplied by the anterior descending branch of the left coronary artery. The degree of myocardial ischemia was evaluated by estimating the exercise tolerance of the animal on a motorized treadmill. Before the cellophane wrap was placed around the coronary artery, the animals would run nine to twelve minutes at 8½ miles per hour on the treadmill before becoming extremely anxious, begin to whine, and salivate prolag on the mill, and would break alternatively from a gallop to a run. Eventually they would become anxious to escape the mill. When the exercise was discontinued, these animals would pant, but would drink water and appear

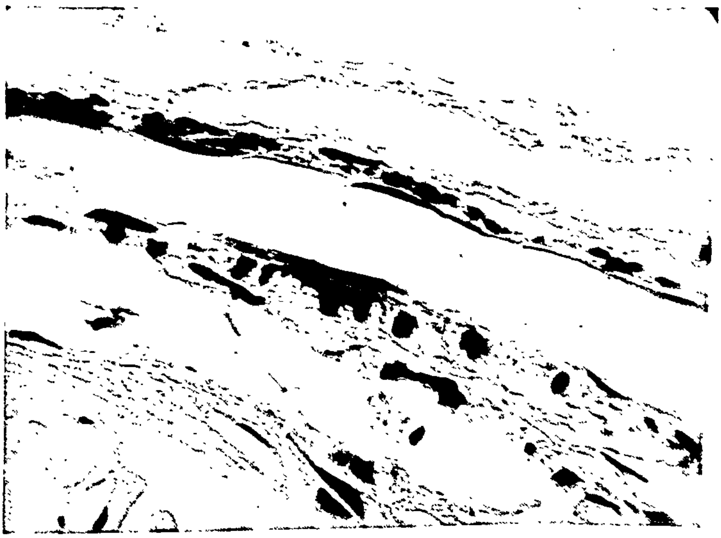


Fig. 8.—High-power view of small branch showing well-developed muscle and elastic tissue in same animal as shown in previous slide at 7 o'clock.

similar to any other normal dog after exercise. Five months after these animals had had a piece of cellophane wrapped around their anterior descending branch of the left coronary artery, they could run for one and six-tenths minutes on the treadmill before becoming extremely anxious, begin to whine, and salivate profusely. If the exercise was not terminated, they would attempt to lie down or drag their feet on the revolving platform. When the mill was stopped, they would drop where they stood and resist all coaxing to move for some minutes and would not drink water. Animals that had reached this stage were then subjected to a left internal mammary artery implant. Four months after the implant those animals which had definitely developed an internal mammary coronary anastomosis had a return of exercise tolerance to seven minutes or more. This occurred in spite of a completely occluded anterior descending branch of the left coronary artery. When an internal mammary coronary anastomosis failed to develop, there was no improvement in exercise tolerance in such animals after implantation. Because of these experimental results it was thought that implantation of the internal mammary artery into the left ventricular myocardium might be of value in the treatment of human cases of coronary artery insufficiency.

## HUMAN CASES OF CORONARY ARTERY INSUFFICIENCY TREATED BY INTERNAL MAMMARY ARTERY TRANSPLANT

*Selection of Cases.*—The estimation of clinical results is always difficult and the results of any given surgical procedure may vary according to the severity of the disease process at the time of operation. There are certain well-known pathologic facts concerning coronary artery sclerosis and thrombosis which greatly influence the selection of cases for internal mammary artery implant. Perhaps the most important is the fact that coronary artery sclerosis in general is confined to the first 3 or 4 cm. of the coronary arteries. It has been stated that the arteries distal to the first 3 or 4 cm. of the coronary vessels show lesser degrees of sclerosis and rarely is sclerosis seen after the 3rd or 4th order of branching.[12, 13] It has been shown that in cases of severe coronary artery sclerosis, the vessels lying within the heart muscle are generally free of arterial disease. Thus, an internal mammary artery placed between ventricular muscle fibers is in an area where the arteries are comparatively healthy. In this way, fresh blood can be brought to the network of nonsclerosed arteries which exist beyond the points of coronary artery obstructions.

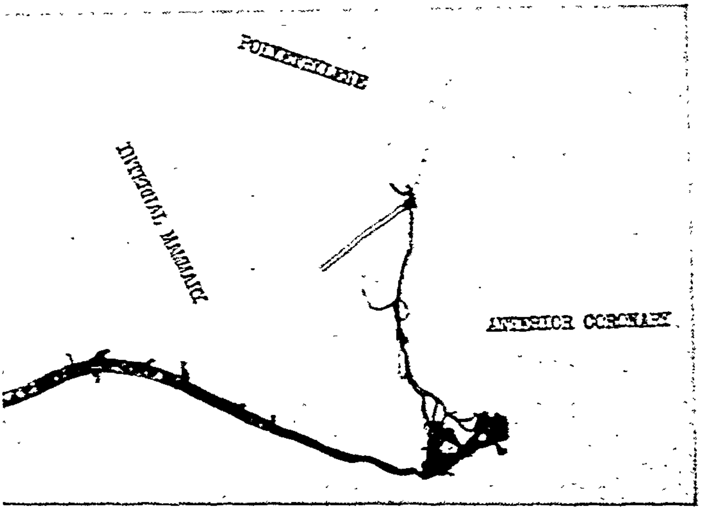


Fig. 9.—Shows a cast made by injecting the implanted internal mammary artery twenty-two weeks after implantation. Note the new branches of the implanted internal mammary artery which join with those of the anterior descending branch of the left coronary artery. A piece of polyethylene tubing was tied around the anterior descending branch at its origin before digestion was started.

In cases of coronary artery thrombosis with myocardial infarct, the picture is entirely different. In these cases, if the patient survives, an area of myocardial muscle undergoes degeneration and eventually heals by scar formation;

and there should be healthy muscle surrounding the area of the healed infarct. It is also stated that during the process of healing new blood vessels grow into the infarcted area. Thus, the history of a left coronary artery thrombosis with recovery does not constitute a contraindication to internal mammary implantation. The new living artery can be placed in healthy muscle which is present at the edge of the healed infarct and, if necessary, into the intraventricular septum itself. With this in mind, it is clear that the patients who have recovered from coronary artery thrombosis and infarction may be considered as candidates for an internal mammary artery transplant. One of our patients was known to have had two attacks of coronary artery thrombosis with posterior infarction. At operation there was still what appeared to be good muscle posteriorly. There was, however, evidence of scarring on the posterior surface towards the apex which scarring extended for half an inch or more on the anterior surface of the left ventricle. The internal mammary artery was placed into healthy muscle just proximal to this scarred area. We have been reluctant to operate upon patients who are able to carry on their normal daily activities. We have, to date, operated only upon those patients who are unable to carry on because of the severity of their anginal pain. Patients with an enlarged left ventricle and myocardial decompensation have been considered poor risks, and have not been accepted. Every attempt has been made to exclude other sources of anginal pain, and to make certain that the pain from which the patient is suffering is due to coronary insufficiency. Where there is a doubt, and particularly when other organic disease such as cholelithisis exists, the associated disease has been treated first.

## CLINICAL RESULTS

We have operated on four human cases suffering from coronary insufficiency.

CASE 1.—The patient, aged 53 years, was admitted to Royal Victoria Hospital on April 24, 1950. He had been unable to work for three years because of substernal pain which radiated to the left jaw and down the left arm to the wrist, and was present day and night. It was first noticed fourteen years prior to admission, gradually becoming more severe. Some, but not complete, relief was obtained by nitroglycerine. Exercise tolerance was limited to one city block; he walked very slowly before onset of anginal pain occurred. There was a history of 40 pounds loss of weight in the past ten years. There was also a history of intolerance to fatty foods with eructations of gas and inability to eat a large meal.

All investigations were negative, except for the electrocardiograms which showed marked coronary insufficiency, accentuated by slightest effort. This was a case of angina status.

On April 28, 1950, the left internal mammary artery was implanted into the left ventricular myocardium. This was carried out through a left transthoracic approach and the sixth intercostal artery was cut and was pulled with the internal mammary artery into a tunnel in the myocardium. Except for a continuously low blood pressure throughout the operation, the patient appeared to tolerate the operation very well. The postoperative convalescence was uneventful until sixty hours after operation, at which time he developed sudden severe substernal pain and died two hours later. The interval between completion of operation and death was approximately sixty-two hours.

*Pathologic Findings* at autopsy revealed that the three main coronary arteries were pipe stem in character. The right coronary artery was completely blocked by an old, firm grayish thrombosis. The left anterior descending branch was completely occluded for a distance of 1 cm. by a recent, soft, dark-brown thrombosis. The internal mammary artery was patent throughout (Fig. 10). There was no hematoma at the site of implant. Death was due to a recent thrombosis of the anterior descending branch of the left coronary artery with ventricular infarction. It is our opinion that had this man's blood pressure been maintained throughout the operation and not been permitted to drop to as low as 70/60, which it was at the time of implantation, that this patient might not have developed a fresh coronary artery thrombosis. The maintenance of blood pressure is particularly important in cases of coronary artery disease. Slowing of the rate of blood flow through a narrowed irregular coronary artery will favor thrombosis.

In our last three patients, blood pressure has been maintained by means of a continuous drip of neosynephrin, which is started at the same time as the anesthetic. In these patients the blood pressure was well maintained throughout the operation. All three patients have had a comparatively uneventful postoperative recovery.

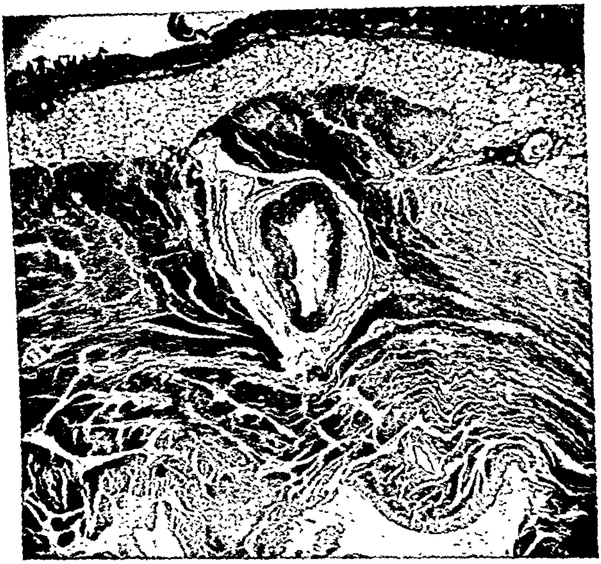


Fig. 10.—Internal Mammary Artery of Case No. 16415; Sixty-two hours after implantation lying within the myocardium. The arterial lumen is free of thrombus. There is little reaction around the artery and there is no hematoma. Note preservation of surrounding myocardial muscle fibers.

Case 2.—The patient was a man 54 years of age who suffered from severe precardial pains which radiated down the left arm, and inability to ingest solids. This pain was brought on by emotion, exercise, or the ingestion of solid food, and prior to operation this patient had lived on a liquid diet for three months and had not worked for nine months. There was dyspnea on slight exertion. Exercise tolerance was limited to half a city block. Electrocardiograms showed coronary artery insufficiency accentuated by exercise; all other investigations were negative. On November 2, 1950, his left internal mammary artery was implanted into the left myocardium. His postoperative course, except for abdominal distention, was uneventful.

The patient left the hospital and flew to his home in the Canadian West at the end of five weeks, and at the present time is free of pain, is working a full day, and able to take solid food without pain.

Case 3.—The third patient, a man 49 years of age, suffered from severe angina pain for four years. The pain started in the substernal region and radiated down the left arm. Occasionally the pain went to the throat and jaw. The pain was made worse by any change in pace, by exercise, or by emotion. Electrocardiograms showed evidence of an old healed posterior wall infarct. He was operated on Nov. 20, 1950. At operation the posterolateral wall, the apex, and half an inch of the anterior wall of the left ventricle were found to be scarred. The internal mammary artery was implanted into healthy muscle proximal to the apical scar. Except for a paralytic ileus, which was easily controlled, the patient's postoperative convalescence was uneventful. He returned to his home at the end of four weeks and was free of pain until Jan. 2, 1951, when he developed pain in the left wrist and forearm. This pain was associated with exercise or emotion. There was no pain in the pectoral region and none in the throat. Electrocardiograms taken in March, 1951, showed continued improvement. His present pain is interpreted as that which sometimes occurs after an anterior wall infarction, and has been compared to the shoulder arm syndrome.

CASE 4.—This man, aged 55 years, suffered from substernal pain for two years which radiated from midsternum down both arms. Pain was brought on by exercise, emotion, and a moderately fair-sized meal. His preoperative electrocardiograms suggested the presence of an old healed anterior wall infarction. His exercise tolerance was poor. On March 1, 1951, he was operated on. A large healed infarct was found on the anterior wall of the left ventricle. The internal mammary artery was implanted into healthy muscle, lateral to the scarred area. The postoperative convalescence has been uneventful.

Abdominal distention which occurred in Cases 2 and 3 seemed to be avoided by early prophylactic decompression of the stomach with Wagensteen suction. The patient returned to his home in Nova Scotia four weeks after operation at which time he was free of pain.

## DISCUSSION

In our experimental work it has been shown that when the internal mammary artery is placed in the ventricular myocardium, it forms new arterial branches which anastomose with the left coronary circulation. When this occurs, the heart is protected against death by infarction following the occlusion of the anterior descending branch of the left coronary artery. It has also been shown that those animals which have survived ligation of the anterior descending branch of the left coronary artery, die or develop infarction when the implanted internal mammary artery is occluded. An internal mammary coronary artery anastomosis has been shown to be of functional value. It has relieved artificially produced coronary artery insufficiency. We would like to point out that the implant procedure is different from other grafting operations. The internal mammary artery is not placed on the surface of the heart but is pulled into position by blunt dissection between the heart muscle fibres. Little damage is done to the heart muscle fibres and practically no reaction occurs around the implanted artery. Branches from the implant reach the deep coronary arteriolar plexus.

It is reasonable to assume on the basis of our experimental work and the pathological facts of coronary artery sclerosis that an internal mammary artery implant may be of value in the treatment of human coronary artery insufficiency. Cases have to be carefully selected and treated by a medical-surgical team, and it is our opinion that the use of quinidine pre- and postoperatively with procaine during the operation is important in preventing ventricular fibrillation. Our first patient, we believe, developed his coronary thrombosis because of the continuous low blood pressure which was present throughout the operation. In order to bring these patients through an intrathoracic operation without further damaging their coronary artery system, we believe that it is necessary to maintain their blood pressures throughout the entire operative procedure. The postoperative care of these cases is fundamentally a medical problem. Unlike other thoracic cases, these patients should not be moved frequently in the first few postoperative days. It would seem best to treat them postoperatively much in the same manner as a case of coronary artery thrombosis. Interpretation of postoperative electrocardiographs is difficult because of the disturbances created by the implantation of a pulsating artery into the anterior wall of the left *myocardium*. Clinically there has been no evidence of coronary artery thrombosis developing after operation in the three cases which have survived.

It is too early to estimate results other than to say that the patient in Case 2 has returned to work and is free of pain. It is now five months since his operation. We believe the operation should have a very low mortality rate. The actual work on the heart takes about two and one-half minutes, and causes the heart no embarrassment. Should the implant fail to form an anastomosis, the patient should be no worse off than before operation.

## SUMMARY

1. The internal mammary artery has been shown experimentally to be capable after implantation of carrying the circulation of the left ventricle.

2. The internal mammary artery has been successfully transplanted into the ventricular myocardium in man. The operative procedure is simple and appears to cause no disturbance to cardiac function.

3. The implanted artery, when it forms an anastomosis, furnishes a third artery to the left ventricle beyond the points of coronary artery occlusion.

4. The internal mammary artery in man, as in the animal, was found to be completely patent sixty-two hours after implantation in the one fatality that occurred.

5. Three of four patients have survived the operation. One is well and working at the end of five months, and the others show improvement. There is no evidence that the procedure causes any harm to the existing coronary circulation.

I wish to express my appreciation for the counsel, criticism, and continued support which have been given by Dr. Gavin Miller, Dr. Lyman Duff, Dr. Donald Webster, and Dr. C. A. MacIntosh. In particular do I wish to thank Dr. G. R. Brow for his careful selection of cases for operation and for his help in their postoperative care.

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## DISCUSSION

DR. SAMUEL A. THOMPSON, New York.—Dr. Vineberg has shown by his experiments that anastomosis between the internal mammary and the coronary blood vessels can be accomplished, and in this way additional vascularization of the myocardium occurs. We have approached the problem of coronary insufficiency in a different manner. We perform cardiopexy by instilling powdered magnesium silicate inside the pericardial sac. This produces a granuloma and causes additional vascularization by extracardiac anastomosis as well as by intercoronary anastomosis. The myocardium is converted from an ischemic condition to one of hyperemia. We have operated upon fifty-two patients, the first one is now thirteen years after operation, and we do not feel that there is any tendency for constrictive pericarditis to develop following this procedure.

I would like to emphasize that this type of treatment is not a cure for coronary disease. It is a method of rehabilitating a group of cardiac cripples. Our operative mortality and hospital mortality has been 12 per cent. Of the surviving patients followed to the present time, or to the time of their death, 85 per cent have been more than 50 per cent improved and rehabilitated. Seventy per cent of them are more than 66 per cent improved, and 20 per cent of them claim that they are completely normal.

I would like to ask Dr. Vineberg if he feels that his method gives vascularization beyond the local area in which the blood vessel is grafted.

DR. VINEBERG (closing).—Yes, Dr. Thompson, I believe that the implanted internal mammary artery is capable of taking over the circulation of the entire left ventricle. We have no evidence that it vascularizes the right ventricle.

Approximately 50 per cent of all cases of coronary artery thromboses involve the left coronary artery and it is in these cases that there is the highest mortality and morbidity. The internal mammary artery implant experimentally forms an anastomosis with the left coronary circulation on an average of 50 per cent. In some series, however, anastomosis has occurred in 100 per cent of the implants. When the anastomosis does form it completely revascularizes the left ventricle, and gives 100 per cent protection against death and infarction, following occlusion of the anterior descending branch of the left coronary artery.

# CONGENITAL CYSTIC DISEASE OF LUNG ASSOCIATED WITH ANOMALOUS ARTERIES

FREDERICK G. KERGIN, M.D., M.S., F.R.C.S.
TORONTO, ONT.

IN 1946 Pryce published the first comprehensive description of a congenital lesion of the lung which has two components, a maldevelopment of part of the bronchial tree to a lower lobe with an associated distribution of arterial blood to the affected portion of the lung by way of an anomalous artery arising from the aorta. In 1947, he, with Sellors and Blair, published a second paper dealing further with the etiological, pathologic, and clinical aspects of the condition. Both papers were based on a study of pathologic material obtained from seven patients who had been treated for this condition by pulmonary lobectomy.

The bronchial abnormality may affect either lower lobe, more commonly the left, and involves the posteromedial portion of the lobe. In this area the bronchial maldevelopment may be of the nature of bronchiectasis of a congenital type or, more commonly, there is a bronchopulmonary mass made of cysts of bronchial origin surrounded by nonfunctioning lung tissue of a primitive type. In some cases the congenital separation of this mass from the bronchial tree appeared to be complete, while in others there seemed to be continuity of developmental origin. In some a secondary connection with the bronchus appeared to have occurred as the result of infection. The symptoms which brought these patients to treatment were due to infection of the bronchopulmonary mass. In those cases which had a pre-existing bronchial connection this could occur by bronchial spread, but in others in which the separation had been complete it was necessary to postulate direct spread of infection from contiguous lung, or hematogenous infection.

In the examples studied by Pryce there was a failure of development of branches of the pulmonary artery to the affected part of the lobe. Instead of venous blood, carried by the pulmonary artery, the bronchopulmonary mass was supplied with arterial blood through an anomalous artery of considerable size arising from the aorta and passing to the lower lobe between the leaves of the pulmonary ligament. This artery may arise either from the lower thoracic aorta or from the abdominal aorta and then pass upward through the diaphragm to enter the ligament. Pryce stated that after passing through branches ramifying over the mass this blood then drains into branches of the inferior pulmonary vein. He pointed out that, although this artery carries oxygenated blood at systemic blood pressure, it has the histologic structure of a pulmonary artery, and is subject to early atheromatous degeneration.

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Read (by title) at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, New Jersey, April 16-18, 1951.

Pryce is impressed by the fact that systemic arteries of this type are sometimes found supplying normally connected and developed lung, and from this and other considerations concluded that the vascular anomaly is primary. He postulated that in the course of development of a lung, the bulbous tip of one branch of the primitive bronchus may come into relationship with and be "captured" by part of the dorsal capillary plexus, which drains into the primitive dorsal aorta. With further development the connection may persist, giving rise to an anomalous artery or arteries arising from the aorta and supplying lung. If this abnormal connection does not lead to a distortion of growth of the affected bronchus, the result is an anomalous artery supplying otherwise normal lung. If, however, the traction resulting from the abnormal connection results in dislocation of the bronchus, some form of congenital bronchial lesion results, again with anomalous arterial connection. If the abnormal mass remains within the lung, the condition under discussion results, which Pryce has called "intralobar sequestration." If the dislocation is more complete, the abnormal lung mass becomes separated from the lobe to form a lower accessory lung, which he has termed "extralobar sequestration." Finally, he suggests that there may be death of the captured bulbous tip, and this results in agenesis of part or all of the affected lung.

Pryce's theory seems to explain the known facts in regard to anomalous vessels connected to lung better than any other, and has the virtue of relating intra- and extralobar sequestration to each other as being differences of degree rather than kind. Case 5, to be described below, is an example of both intra- and extralobar sequestration in relation to the same lower lobe, which lends some support to the theory. This anomaly apparently has not been described before.

In 1950 Bruwer, Clagett, and McDonald of the Mayo Clinic reported five cases. They expressed agreement with Pryce's views in regard to the embryological error responsible for the anomaly. In their review of the literature they had found reports of twenty-six similar cases, and mentioned that in three of these there had been a fatality because of hemorrhage from the anomalous vessel.

The present report is based on five examples of this condition which have been encountered at the Toronto General Hospital. In the earlier records there are three other cases of localized congenital cystic disease of the lower lobe which seem to represent examples of the same condition, but as there is no clear cut description of the anomalous artery, they are excluded from the series.

## CASE REPORTS

CASE 1.—J. S., a man, aged 17 years, was admitted on Sept. 17, 1941. He was perfectly well until six months before admission, when he developed pneumonia. He continued to have a cough, dry at first and later productive of a small amount of mucoid sputum. On one occasion he had a small hemoptysis of bright blood. Examination showed slight diminution in lung resonance and diminished breath sounds at the left base.

Roentgenograms (Fig. 1) demonstrated three ovoid cystic spaces in the left lower lobe, situated posteriorly. One of them showed a fluid level. A diagnosis of cystic disease of the lower lobe was made.

At operation (by Dr. R. M. Janes) there were no pleural adhesions. There was no pulmonary ligament of the usual type, the lung was directly applied to the aorta and esophagus. An artery 6 mm. in diameter passed directly forward from the aorta into the lower portion of the lower lobe. Dissection lobectomy was done.

Examination of the lobe showed that the postero-medial portion contained a firm, rubbery mass, in which individual rounded cysts could be felt. This part of the lung was yellow-pink in color, and devoid of pigment. On cutting the lobe the cysts were filled with inspissated mucus, except one which had drained into the lower lobe bronchus. The cysts were lined by typical bronchial epithelium.

The patient made a good recovery from the operation and has remained perfectly well.

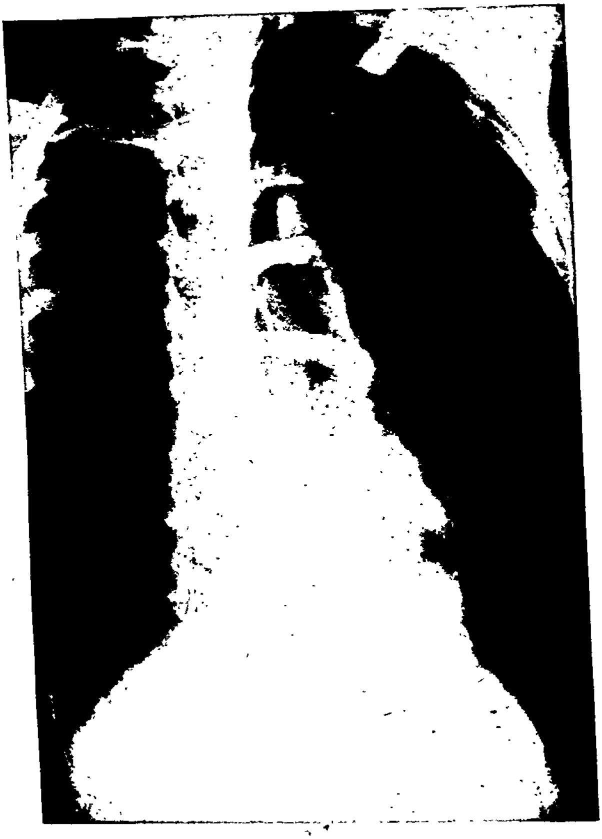
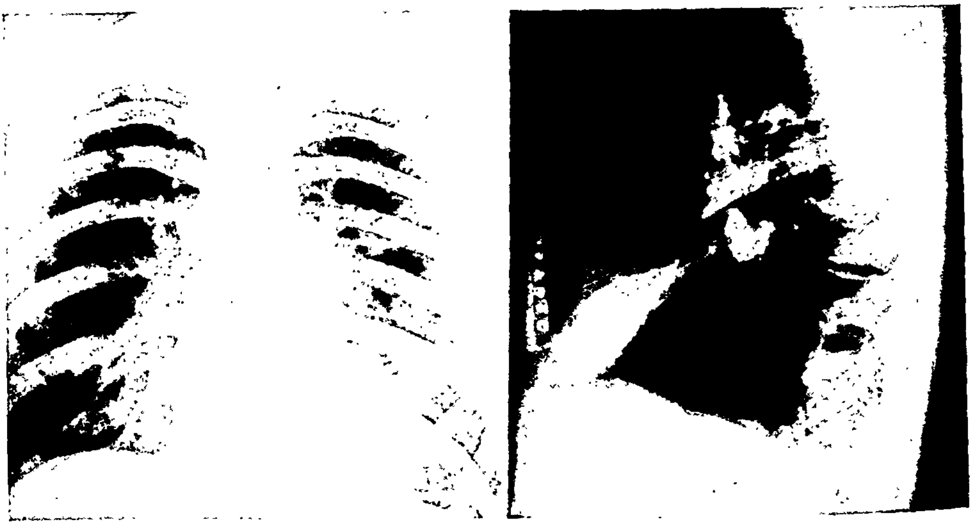


Fig. 1.—Case 1. Oblique view to show rounded density behind heart. In the original film a faint fluid level is visible close to vertebrae.

Case 2.—H. Z., a woman, aged 19 years, was admitted to the hospital on Oct. 12, 1948, with a history of pneumonia on the left side at the ages of 10, 12, and 15 years, and again in January, 1948. At the age of 10 years the left chest had been drained by a tube for a few weeks. Since the pneumonia in 1948 she had had two ounces of green sputum per day. Examination showed diminished breath sounds at the left base posteriorly and the scar of the previous drainage.

Roentgenograms (Fig. 2) demonstrated a large cystic area in the left lower lobe posteriorly, with two fluid levels. A diagnosis of infected bronchogenic cyst was made.



*A.* *B.*

Fig. 4,*A*.—Case 4. Posteroanterior view shows rounded area at left base with fluid level.

Fig. 4,*B*.—Case 4. Lateral view demonstrating cyst with fluid level above, and multiple rounded translucent areas below.

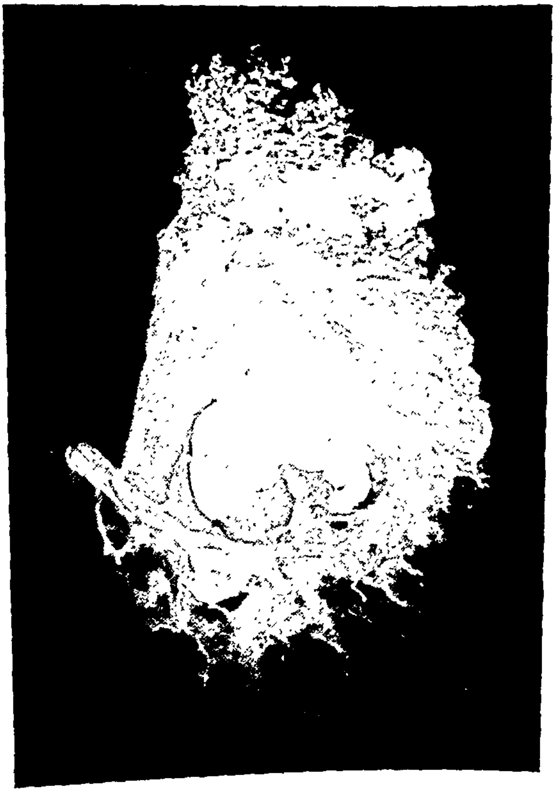


Fig. 5.—Case 4. Roentgenogram of lower lobe after bronchus, pulmonary artery, and anomalous vessel had been injected with plastic. Note translucent areas in lower part of specimen indicating mucus-containing cysts, not communicating with bronchus.

In the laboratory it was found that saline injected into the anomalous artery drained freely from the pulmonary vein. Plastic of different colors was injected into the bronchial tree, the pulmonary artery and the anomalous artery, and then after roentgenograms of the specimen were made (Fig. 5) the lobe was macerated to leave a plastic cast of these structures (Fig. 6). This demonstrated the size and distribution of the anomalous vessel, and also that the pulmonary artery did not distribute branches to the cystic mass. The lower,

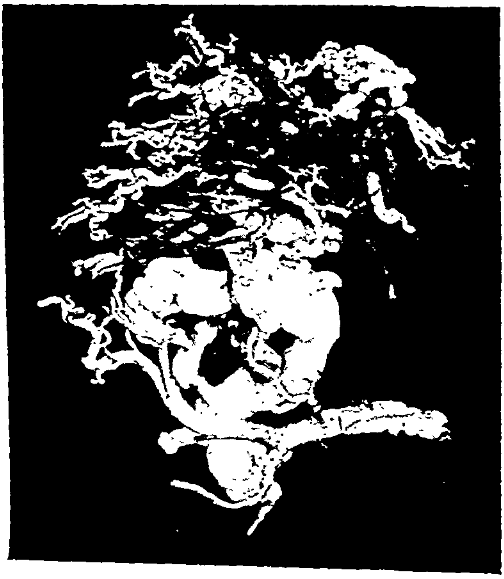


Fig. 6.—Case 4. Cast obtained after maceration of specimen. Large anomalous artery below on right, distributed only to cystic portion of lobe.

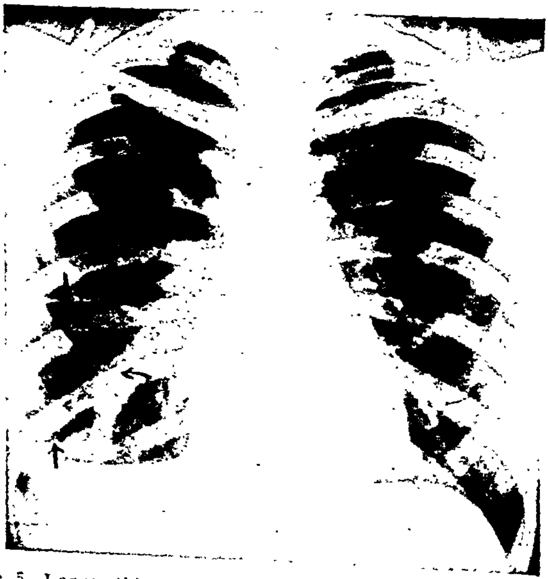


Fig. 7.—Case 5. Large, thin-walled cyst with fluid level, right lower lung field.

rubbery part of the mass was composed of multiple small mucus-containing cysts which were lined by ciliated pseudo-stratified columnar epithelium.

The patient made an uneventful recovery from operation.

CASE 5.—L. M., a girl, aged 16 years, was admitted to the hospital on Feb. 2, 1950. She had pneumonia at the age of 18 months, was said to have had whooping cough 3 times in childhood, had a chronic cough from 2 until 4 years. She was then well until February, 1949, *when she had pneumonia in the right lung, and this recurred in October, 1949, and* in January, 1950. She raised about one-half ounce of mucopurulent sputum per day, but had not had hemoptysis. On examination she was in good general condition; there was slight dullness with decreased breath sounds at the right base posteriorly.

Roentgenograms (Fig. 7) showed a large thin walled cyst with a fluid level in the right base posterolaterally.

A diagnosis of intralobar sequestration of the right lower lobe was made.

At operation the lower lobe was adherent to the parietal pleura. The posterolateral portion of the lobe was filled by a cyst, 8 cm. in diameter, and this part of the lung was yellow-pink in color and devoid of pigment. Just posterior to this area was a rubbery ovoid structure, of similar color, 5 cm. in length, connected to the surface of the lobe by a short, thin pedicle. Passing to this appendage was an artery 3 mm. in diameter rising from the seventh intercostal artery. There was no accompanying vein. No other anomalous artery was found entering the lower lobe. Dissection lobectomy was done.

Examination of the specimen (Fig. 8) showed that the large intralobar cyst was trabeculated, had multiple bronchial connections, and was lined by ciliated pseudostratified epithelium. The appendage was made up of many small cysts containing thick mucus, and the walls contained bronchial elements. Sections of the pedicle demonstrated that there was no bronchial connection with the lung, but the pedicle was traversed by multiple large vessels. It is considered that this is an example of both extra and intralobar sequestration in which the blood supply to the accessory lung was derived from an intercostal artery and in the absence of an accompanying vein passed on through the pedicle to supply the intralobar sequestration; thence draining into the pulmonary vein.

The patient made a good recovery from the operation and has been relieved of her symptoms.

## SUMMARY OF CASES

These five patients ranged in age from 16 to 60 years. All complained of chronic cough with sputum, frankly purulent in 4, foul in 1, and 3 patients had had hemoptysis. Four patients showed an abnormality on examination of the chest, usually decreased percussion note, with diminished breath sounds. No patient showed clubbing of the fingers. In all, one or more cysts communicated with the bronchus; in every case the roentgenogram showed a cyst with a fluid level. In three the left lower lobe was involved, and in two the right lower lobe. In two cases the anomalous artery arose below the diaphragm, in two it arose from the thoracic aorta, and in one patient it arose from an intercostal artery. At operation all patients showed a very characteristic picture of a mass of yellowish-pink, nonpigmented lung contained within the posterior part of the lower lobe, rubbery in consistency, and containing one or more cysts.

After clinical and radiographic investigation, the diagnosis in the first case was cystic disease, in the second bronchogenic cyst. In the third case, the man aged 60 years, a diagnosis of intralobar sequestration was considered, but because the symptoms had come on at 55 years, chronic lung abscess was thought to be a more likely diagnosis. In the last two cases a diagnosis of intralobar sequestration with anomalous blood supply was made with confidence.

All five patients have had a satisfactory result from surgical treatment.

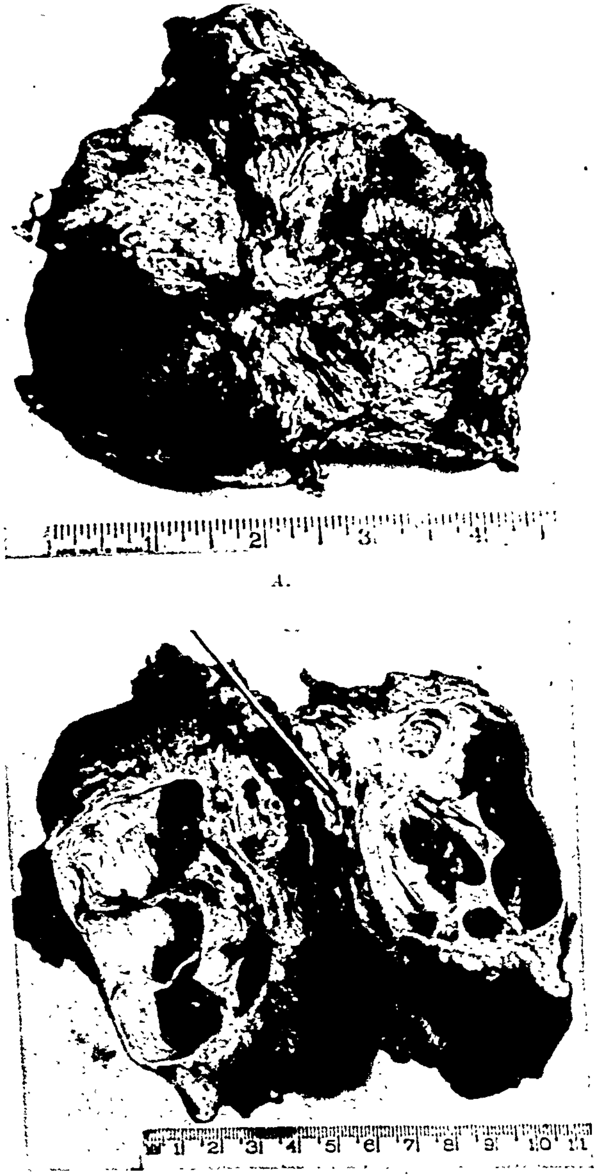


Fig. 8,*A*.—Case 5. Specimen viewed from behind showing lower accessory lung attached to lobe.

Fig. 8,*B*.—Case 5. Lobe opened to show large cyst.

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Five cases of this condition, all treated by lobectomy, are reported. One of these cases showed an unusual association of both intra- and extralobar sequestration in relationship to the same lower lobe.

Observations which tend to prove that circulation through the anomalous artery constitutes an unusual type of shunt are described.

It is suggested that the clinical and radiographic features of this condition are sufficiently typical to permit a correct preoperative diagnosis. The pathologic condition found at operation is usually recognizable with ease.

Excision of the diseased segment or lobe is a satisfactory method of treatment.

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## DISCUSSION

As pointed out by Pryce and also by Bruwer and associates this condition is sufficiently common that it is of some importance to physicians and surgeons interested in pulmonary disease. The pathologic anatomy is such that once infection becomes established, it tends to become persistent and lead to chronic disability. Only excision of the diseased segment or lobe is likely to lead to permanent relief of symptoms. A study of these five lower lobes suggests that in only one (Case 5) the bronchial connection with the cyst was primary; in the other four it was probably secondary.

Pryce stated that the oxygenated blood from the anomalous artery drains into the pulmonary vein, and our observations by dissection of the specimens and by perfusion of the artery support this view. Bruwer et al. stated that the vascular connection is with the pulmonary artery. They suggested that further information might be gained by filling the two vascular systems with a plastic solution and then digesting the lung away to leave a plastic model. The same thought had occurred to us, and Fig. 6 illustrates the model obtained. It demonstrates that the distribution of the two arteries is independent, each confined to its own section of the lobe, but from neither source did the plastic pass through the finer ramifications to reach the pulmonary vein, although saline passed through freely when injected from either the pulmonary or systemic artery.

If the observation that the branches of the anomalous artery connect with the pulmonary vein is correct there is a pathway for an unusual type of vascular shunt within the arterial side of the circulation. The size of the artery, the difference in pressure between the aorta and the pulmonary vein, and the ease with which saline can be perfused suggest that the rate and volume of blood flow through this shunt is considerable. This imposes added work upon the left ventricle and may be of some significance in the older patients. The decided improvement in exercise tolerance in the man of 60 years, in spite of the loss of some functioning lung tissue, may have been due to the interruption of this shunt. His lesion was unusual in that there were two arteries coming from the aorta with one accompanying vein draining into the azygos vein, but as the vein was no larger than either of the arteries it is probable that there was also a connection with the pulmonary vein.

Pryce mentions that in extralobar sequestration the blood from the accessory lung commonly drains into a vein connecting with the azygos vein. The discovery of such a vein in the above case of intralobar sequestration is some evidence that intra- and extralobar sequestration have a similar embryological origin and differ only in degree. Case 5, in which both conditions were found in relation to the same lower lobe, also adds some weight to this view.

The clinical and radiographic features of this condition are sufficiently characteristic that the correct diagnosis was made before operation in the last two of these five cases. The appearance of the lung at operation is striking and easily recognized in most instances, which is fortunate, because the operator can then proceed with caution and by demonstrating the anomalous artery avoid the tragedy which has followed inadvertent division of it on three occasions.

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The clinical and radiographic features of this condition are sufficiently characteristic that the correct diagnosis was made before operation in the last two of these five cases. The appearance of the lung at operation is striking and easily recognized in most instances, which is fortunate, because the operator can then proceed with caution and by demonstrating the anomalous artery avoid the tragedy which has followed inadvertent division of it on three occasions.

## SUMMARY

The etiology and pathology of intralobar sequestration of lung as described by Pryce have been reviewed.

Five cases of this condition, all treated by lobectomy, are reported. One of these cases showed an unusual association of both intra- and extralobar sequestration in relationship to the same lower lobe.

Observations which tend to prove that circulation through the anomalous artery constitutes an unusual type of shunt are described.

It is suggested that the clinical and radiographic features of this condition are sufficiently typical to permit a correct preoperative diagnosis. The pathologic condition found at operation is usually recognizable with ease.

Excision of the diseased segment or lobe is a satisfactory method of treatment.

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# THE PHYSIOLOGY AND CYTOLOGY OF PULMONARY EDEMA AND PLEURAL EFFUSION PRODUCED IN RATS BY ALPHA-NAPHTHYL THIOUREA (ANTU)*

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CLINICIANS and pathologists have observed the appearance of fluid in the lungs and pleural cavities under a great variety of pathologic conditions. Yet despite its common occurrence and great importance, our understanding of the physiology and cytology of pulmonary edema and pleural effusion is still quite limited.

In patients, pulmonary edema and pleural effusion rarely occur unless associated with some complex disease, most notably congestive heart failure, tuberculosis, or pneumonia. Because of this complexity, and because of the critical nature of these diseases, it is almost impossible to develop quantitative methods for studying edema and effusion in the lungs and pleural cavities of living patients. Experimental work on animals, therefore, at present remains our best possible source of knowledge.

For animal work we must have a simple means of producing pulmonary edema and pleural effusion with regularity, yet without doing damage to, or involving any other system of the body; also we must have accurate methods of measuring the amounts of pulmonary and pleural fluids.

These two requirements are met to some extent at least by the use of the "toxic thioureas."[1, 2] Methods of measuring the fluids have now been worked out for the Norway rat.

The "toxic thioureas," of which alpha-naphthyl thiourea (ANTU) has been most extensively studied, are highly toxic for the Norway rat and somewhat less toxic for the dog, domestic pig, cat, and mouse.[3] In all of these species they produced marked pulmonary edema and pleural effusion with great regularity. It is of special interest for the present purposes that they do not produce ascites or edema in any other organ.

The pulmonary edema produced by the thioureas apparently results from a temporary increase in permeability of the lung capillaries. Since the effects are reversible, these drugs apparently produce no lasting damage to the capillaries.[4] In composition, the fluid in the lungs and pleural cavities closely resembles blood plasma; it has a protein content of 3 to 5 per cent and does not contain any red cells.[5]

To study quantitatively the "pure culture" of pulmonary edema and pleural effusion, the ideal method would be to make continuous records on

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individual animals of the amounts of fluid that appear in the lungs and pleural cavities. Since this obviously is not possible, recourse has to be taken to another method: that of killing animals at definite intervals after poisoning, weighing their lungs, and measuring the amounts of fluid in the pleural cavities. This procedure, involving the use of a large number of animals, makes the Norway rat the animal of choice because it is so readily available and relatively expendable.

The apparently simple task of measuring pulmonary edema from lung weights proved to be a complicated undertaking. We believe that after many mistakes, we have now devised a simple, reliable, standard technique. A report[6] in 1947 gave the results obtained with an earlier technique.* The present report contains the results of our most recent studies for which the revised and improved techniques were used. The emphasis of these studies has been on the understanding of the formation of pulmonary edema and pleural effusion.

### METHODS

A total of 203 adult domesticated rats from our colony and from the Albino Farms, Rutland, Vt., were used for these experiments. With few exceptions, rats weighing between 200 to 300 Gm. were used, since they were known to yield the most constant results. A total of ninety rats were used as normal controls. Many hundred wild and domesticated Norway rats of all ages were used in the preliminary experiments.

It proved necessary to use rats that were essentially free of lung infections (abscesses) and areas of pulmonary consolidation. Although the rats of our own colony had been exposed to a serious epizootic of Type II pneumococcal pneumonia two years before,[7] they proved to have very healthy lungs, only rarely showing any areas of consolidation (less than 1 per cent of the rats). The rats from the Albino Farms also only rarely showed areas of consolidation or abscesses. Rats from several other colonies could not be used because of their defective lungs; in some colonies as many as 50 to 75 per cent showed consolidation.

For two to four days before the experiments, the rats were placed in individual cages, with access to our stock diet† and water ad libitum. They were protected from avoidable noises and disturbances in a room separate from the rest of the colony. Care was exerted to avoid drafts or abrupt changes in environmental temperature.

Alpha-naphthyl thiourea (du Pont) was used for all of these experiments. It has an LD50 of 4.1 mg. per kilogram for domesticated Norway rats of our colony.[5] The ANTU was administered in five different doses: two sublethal doses, 1 and 3 mg. per kilogram, both of which fail to kill any animals; 5, 10, and 50 mg. per kilogram, all three of which kill 100 per cent. The drug was administered intraperitoneally in olive oil suspension, since it is practically insoluble in water. For all dosages each rat received 1 c.c. of olive oil suspension per 100 Gm. body weight.

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*These experiments were started during the war in an attempt to devise an antidote for ANTU poisoning in as much as ANTU was being widely used as a rodenticide.

†Graham flour 72.5 per cent, casein 10.0 per cent, skim milk powder 10.0 per cent, butter 5.0 per cent, calcium carbonate 1.5 per cent, and sodium chloride 1.0 per cent.

To obtain quantitatively reproducible results it was found to be of prime importance to grind the ANTU to very fine particle size before mixing it with the oil and to stir the suspension while injecting the rats. To speed up the injection procedure and thus minimize the rat's excitement, the drug was injected intraperitoneally through an 18-gauge needle, the smallest bore through which the preparation would flow freely.

The following standardized procedure was used in determining the extent of the pulmonary edema and amounts of fluid in the pleural cavities. Since the success of the procedure depends on a number of different operations, the various steps will be described in detail. The rats are etherized in as quick and uniform a fashion as possible. For this purpose a quart Mason jar is used in which the rats can readily be observed at all times; and accordingly, can be removed each time at approximately the same stage of anesthesia. Holding the rats by their haunches during the entire process shortens the time of etherization and greatly reduces the severity of the animal's struggle against the ether.

Once anesthetized, the rats are removed and tied down on their backs on a small operating board. More ether is administered by the open drop method throughout the following procedures:

1. A wide strip of skin is cut away with scissors from the pubic arch to the clavicle and an incision is made into the abdominal cavity from the pubic arch to the sternum, care being taken to avoid entering the pleural cavities.

2. With the first and second fingers of the left hand the viscera are pulled to the side exposing a large free area over the vena cava and abdominal aorta. This area is further exposed by retracting the wall of the abdomen to the opposite side with a fixed hook.

3. The abdominal cavity is checked for the presence of unabsorbed olive oil and for any signs of incidental disease or ascites, and any oil present in the exposed area is thoroughly removed by swabbing. Enough of the vena cava is then freed of fat and peritoneum to provide unobstructed access to the vessel.

4. A 20-gauge needle, attached to a 1 c.c. tuberculin insulin syringe, is introduced into the vena cava and 0.1 c.c. per 100 Gm. of body weight of a solution of heparin sodium (1 c.c. = 10 mg.) is slowly injected to prevent coagulation of the blood.

5. A tear is purposely made in the wall of the vena cava in removing the needle thus allowing the blood to flow freely. All the blood is mopped up with weighed cotton swabs until the flow becomes very sluggish. Then the abdominal aorta is cut across with a pair of scissors and the blood is mopped up again until the flow ceases. The collected swabs are then weighed. This entire process requires about five minutes. The rats usually continue to breathe until well after the aorta has been severed; occasionally they stop breathing before section of the aorta and then start breathing again afterward. Six swabs, each weighing 200 to 250 mg., are used for mopping up the blood.

When blood is desired for hematocrit or cytological studies it is withdrawn from the vena cava through the needle used for introducing the heparin sodium. The tuberculin syringe is removed from the needle and replaced with a 10 c.c. syringe. As much as 12 c.c. of blood may be withdrawn in this way. The bleeding process then continues as described above.

6. The thoracic cavity is entered with care to prevent the loss of fluid into the abdominal cavity. A cut through the ribs is made about 1 centimeter to the right of the midline and continued up to the clavicle. The diaphragm is cut just under the sternum and the ribs are cut from the xiphoid process to the clavicle about 1 cm. to the left of the midline. The sternal flap thus formed is reflected over the animal's head and anchored with a hemostat.

7. Immediately thereafter a curved hemostat is placed just under the heart, occluding all of the cardiac vessels and preventing the flow of the little remaining blood from the heart to the lungs.

8. All of the walls of the thoracic cavity, including the sternal flap, are swabbed in a uniform manner three different times, each time with a new, weighed cotton swab (about ¼ cm. in diameter). These swabs are then weighed to determine the amount of fluid present. When the cavity contains enough fluid to be aspirated, as much fluid as possible is removed with a syringe prior to the swabbing.

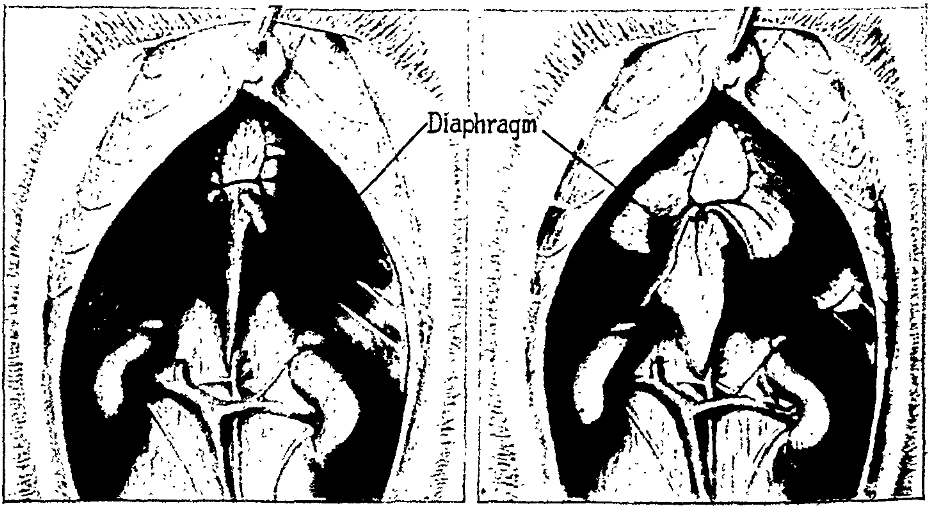
9. The left lung is dissected free and inspected for signs of edema or consolidation. Next a ligature (silk braid) is looped around the lung and tied tightly at the hilum; a cut is made with a pair of fine scissors just proximal to the ligature and the ends of the silk braid are cut at the knot. The surface of the lung is dried lightly on blotting paper before the organ is weighed.

The same process is repeated with the right lung. Owing to its multilobular structure the right lung is more difficult to free. It normally weighs almost twice as much as the left lung.[9]

The pleural effusion swabs are weighed on a torsion balance sensitive to 0.1 mg.; the swabs for the blood weights and lungs are weighed on a balance sensitive to 10 mg.; matched watch glasses are used for the swabs and lungs.

The chief difference between this procedure and that used in our earlier studies lies in the method by which the rats were killed. Formerly the rats were killed by crushing the cervical cord with a pair of pliers. This method had a marked effect on the lung weights and consequently did not permit accurate measure of the fluid in the lungs. In the first place, crushing the cervical cord caused prolonged convulsions in some animals and only mild convulsions in others. Consequently in some rats more blood was forced into the lungs than in others, either as a result of the force of the convulsion or of the longer time interval during which the heart could pump more blood into the lungs. Owing to the varying amounts of blood in the lungs it was never possible to obtain an accurate measure of the amounts of fluid that the lungs contained. In some instances, when crushing the cervical cord caused intracranial and oral bleeding, blood was actually aspirated into the lungs.

Allowing the rats to bleed to death makes it possible to remove all except very small amounts of blood from the lungs. Furthermore, this procedure is carried out on a schedule thus ensuring much more uniform treatment of each rat. A special advantage of the described technique is that opening the chest no longer causes bleeding so that contamination of the pleural fluid with blood is avoided. The use of the ligature of the hilum of the lung prevents the escape of fluid and so further contributes to the accuracy of the procedure.



*A.* *B.*

Fig. 1.—Drawing of typical eviscerated rats: (*A*) showing the diaphragm of normal rat; (*B*) the diaphragm of a rat that had been poisoned sixteen hours before with ANTU.

## RESULTS

### A. PLEURAL EFFUSION

Fig. 1 shows the diaphragm of a normal rat and of a rat killed sixteen hours after having received a lethal dose of ANTU (50 mg. per kilogram). The drawings were made just after the rats had been killed and all the intraperitoneal viscera removed. The diaphragm of the normal rat is concave and for the most part in close contact with the walls of the thorax. In marked contrast, the diaphragm of the poisoned rat bulges far down into the abdominal cavity, literally forming a bag of fluid.

The drawing of the sagittal sections in Fig. 2 shows the distribution of fluid in the intact animal when standing in its normal posture. Immediately after the rats were killed with ether, a string was tied tightly around their necks to avoid any changes in the relative position of the organs that might follow the collapse of the lungs and the resulting escape of either air or fluid. Then the rats were placed in a deep-freeze unit, where they were supported in their normal standing posture by straps, and kept there until they were thoroughly frozen in order to prevent any other post-mortem changes.

After freezing, sagittal and transverse sections were cut with a small, thin-bladded bow saw. Wiping the cut surfaces with a wet cloth brought out the outlines of all of the internal viscera in clear detail.

In Fig. 2 the drawing at the left shows a midline sagittal section of a normal healthy rat. The lungs are in close contact with the heart and diaphragm and so fill the entire thoracic cavity. The pleural space is not visible. The drawing at the right shows a midline section of a rat that had received an intraperitoneal injection of ANTU (50 mg. per kilogram) sixteen hours before being killed. Fluid (solid black areas) fills the greatly distended pleural cavity. The diaphragm is pushed down and away from the heart. The lungs float in the fluid and appear to be collapsed. It is clear that the volume of the fluid in the poisoned rats many times surpasses the volume of the lungs, so that the air space must have been greatly reduced.

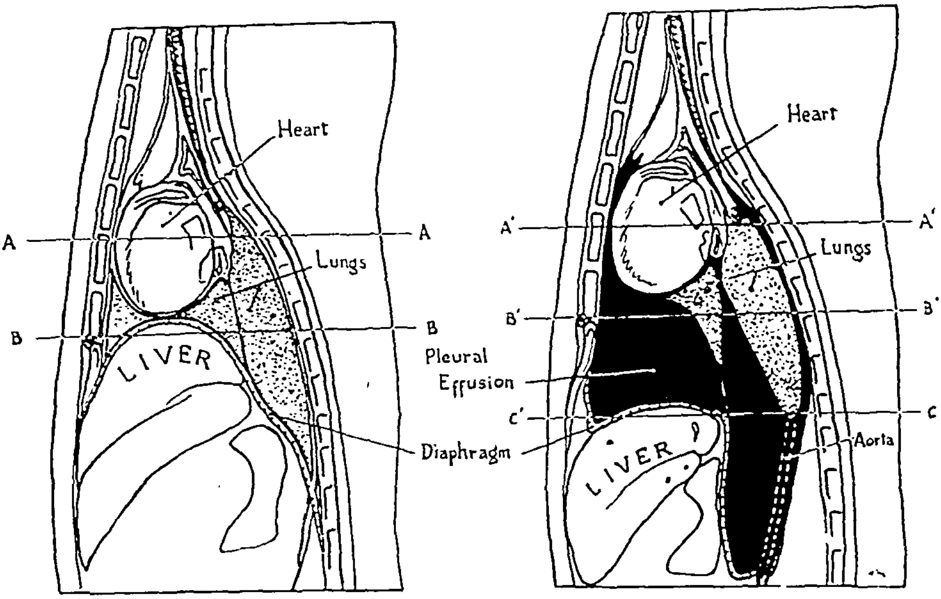


Fig. 2.—Drawing made of typical sagittal sections through the midline of rats frozen in horizontal position in a deep freeze: left, from a normal rat; right, from a rat poisoned sixteen hours before with ANTU.

*Normal Rats.*—

*Pleural Fluid and Body Weight:* Fig. 3 shows the amounts of pleural fluid found in eighty-three normal (untreated) rats, as related to their body weights. The ordinates give the pleural effusion in per cent of body weight, the abscissas body weight in grams. Each dot represents one animal. The solid line is drawn through points obtained by taking the average in each 50-gram interval and represents the proportion of pleural effusion with increasing weight. This remained essentially the same for all rats weighing between 100 and 350 Gm., not surpassing 0.1 per cent in any case. As might be anticipated, the relative amount of effusion was greater in very young animals, averaging 0.12 per cent for weights up to 50 Gm. and 0.1 per cent of body weight between 50 and 100 Gm. Thus, the amount of fluid in the pleural cavity of normal animals is very small, having a mean of 0.05 per cent of body weight for an adult rat, and a standard deviation of 0.024.

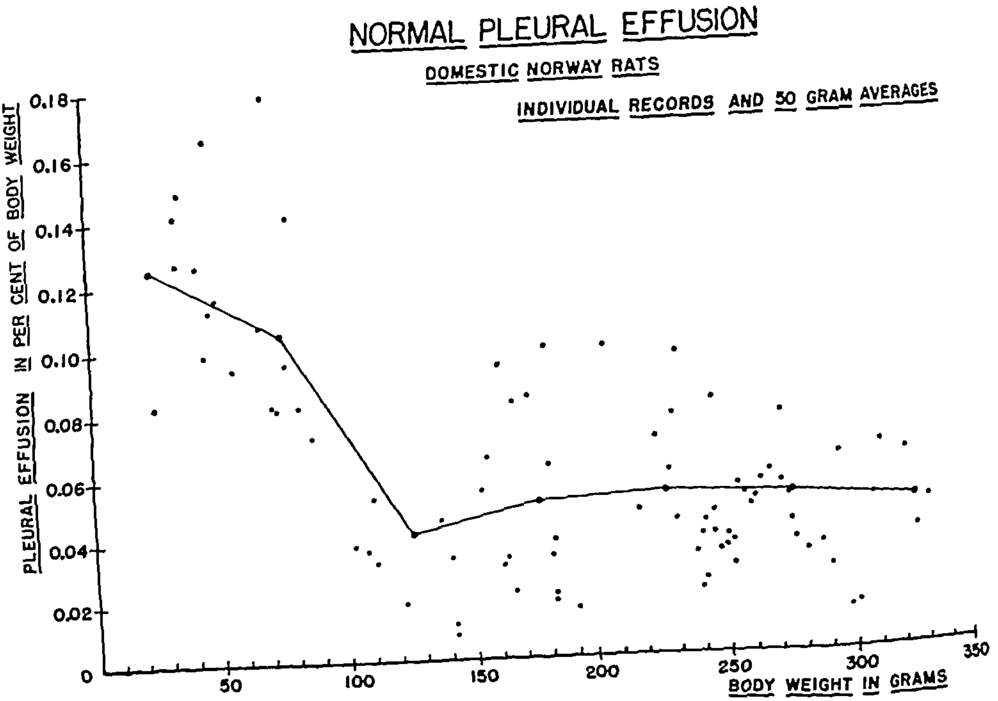


Fig. 3.—Chart showing the amount of fluid in the pleural cavities of individual normal rats at various body weights. Ordinates represent the fluid in per cent of body weight; the abscissas body weight in grams. Each dot represents an individual rat. The solid line connects averages for each 50-gram weight interval.

*ANTU Poisoned Rats.*—

*Onset and Rate of Development of Pleural Effusion:* Fig. 4 shows pleural effusion curves for the different dosages of ANTU. The ordinates give the pleural effusion in per cent of body weight; the abscissas time in hours after the ANTU injection. The curves show the averages for one-hour intervals. During the first hour the averages did not increase above the normal 0.05 per cent levels shown in Fig. 3. During the second hour the amount of pleural effusion increased for all except the 1 mg. per kilogram dosage. Thereafter the amount of pleural effusion increased at an almost constant rate for the 3, 5, 10, and 50 mg. per kilogram dosages, reaching an approximate level of 3.5 per cent during the seven to eight hour interval. The 1 mg. per kilogram dosage did not produce an increase in pleural effusion at any time.

Owing to the high rate of mortality of the rats on the 5, 10, and 50 mg. per kilogram dosage after the eighth hour, observations after longer periods of poisoning with these three doses could be made on only a few rats. In our earlier experiments the pleural effusion reached 5.0 per cent in a number of animals that survived eighteen hours, and 6.3 per cent in one rat that was killed nineteen and one-half hours after having received a 10 mg. per kilogram injection of ANTU.

It is noteworthy that in rats which had received the higher dosages of ANTU and had survived seven to eight hours or more, the thoracic cage appeared to be markedly distended, in some instances literally bulging out from the rest of the body.

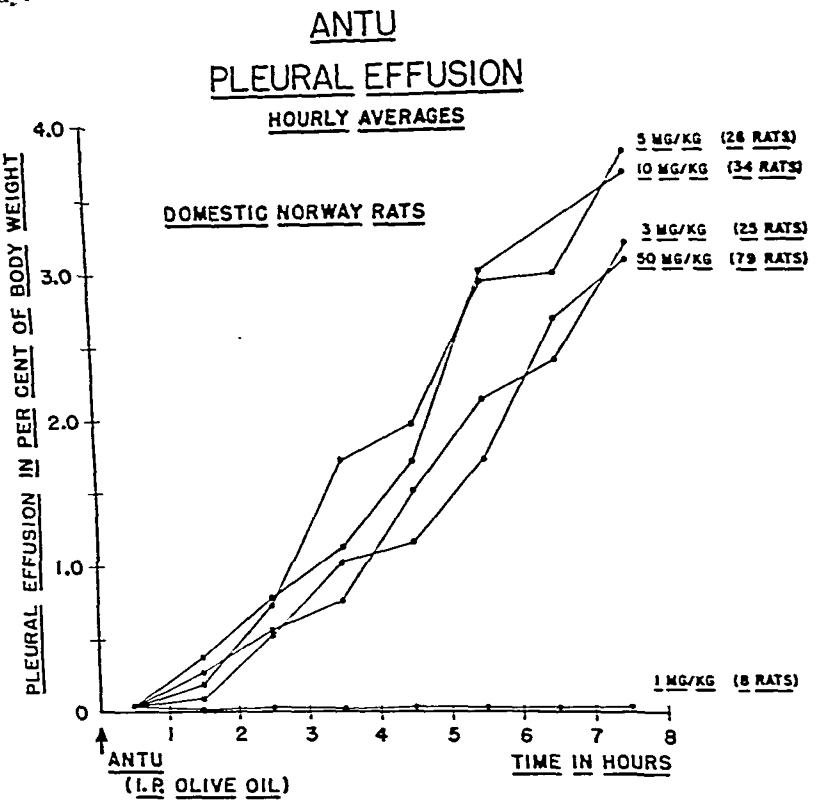


Fig. 4.—Curves showing average amounts of pleural effusion found at hourly intervals after poisoning with five different doses of ANTU. Ordinates represent pleural effusion in per cent of body weight; abscissas time in hours after ANTU was injected.

Since the pleural fluid averages 0.05 per cent of the body weight in normal animals, the high amounts found in the ANTU poisoned rats, 5 to 6 per cent of body weight, represented a hundred-fold increase. The magnitude of the increase can be visualized in another way. In normal rats the lungs occupy a thoracic space that measurement of twenty adult animals showed to average 3 cubic centimeters. In ANTU poisoned animals the maximum amount of pleural effusion occupied 12 to 15 cubic centimeters, a three- to fourfold increase in the thoracic space. Thus, the pleural cavity is capable of a great increase in volume that is probably largely accounted for by a lowering of the diaphragm, but also to some extent to the distention of the thoracic cage. In ANTU poisoned rats the diaphragm apparently is stretched to its full extent.

*Duration of Pleural Effusion:* Duration of the pleural effusion was studied by the use of a dose that produced marked pleural effusion without killing the rats. The 3 mg. per kilogram dose proved to be very satisfactory for this purpose.

Fig. 5 (top record) shows the individual pleural effusion volume of fifty-two rats sacrificed at varying intervals after administration of a 3 mg. per kilogram dose. The pleural effusion first appeared just shortly after the end of the first hour and had established a fairly constant rate of increase by the end of the second hour. It reached its highest point on the eleventh and fourteenth hours. After the fourteenth hour the amounts of pleural effusion definitely decreased. For the next twelve hours some rats still showed moderate amounts, others scarcely any at all. Five rats examined at forty-four to forty-six hours showed only normal amounts of pleural effusion.

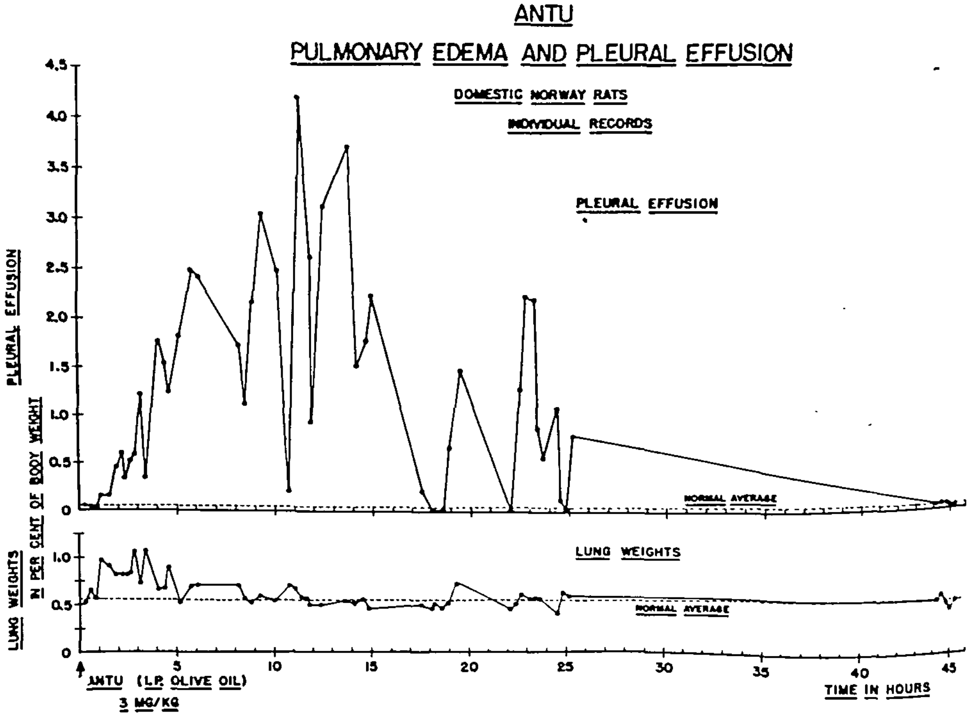


Fig. 5.—Curves showing the pleural effusion and lung weight records for individual animals that received ANTU, 3 mg. per kilogram in olive oil by intraperitoneal injection. Each dot represents an individual rat. The dotted lines give the averages for pleural effusion and lung weights for normal rats ranging in weight from 200 to 300 grams.

It is noteworthy that during the first fourteen hours the pleural effusion would appear to have increased at the same rate in twenty-three of twenty-six rats. The pleural effusion of the rat examined on the fourteenth hour amounted to 3.7 per cent of the body weight. Statistically, then, it would seem very likely that almost all of the rats examined at later times, would, if they had been examined during the first fourteen-hour period, have shown equally large amounts of fluid. The fact that many of them showed less fluid, eleven almost none at all, would indicate that during various intervals after the fourteenth hour the pleural fluid must have been resorbed. In some instances the fluid may have been resorbed in only a few hours.

These results agree with the observation recently reported by Courtice and Simmonds.[10] These workers found that homologous dye-plasma introduced into the pleural cavities of rabbits is absorbed at a very rapid rate. These workers concluded from observations on cats that all of the plasma fluid in the pleural cavities is absorbed through the right and left lymphatic ducts.

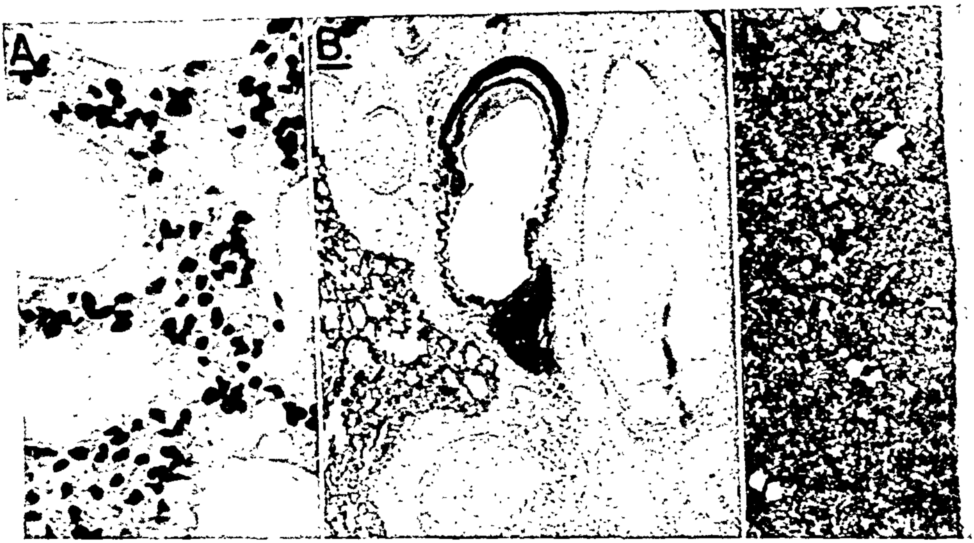


Fig. 6.—Photograph of sections made of the lungs of ANTU poisoned rats. (*A*) lung of rat killed four hours after poisoning with ANTU, 50 mg. per kilogram; (*B*) lung of rat killed eighteen hours after poisoning with ANTU, 50 mg. per kilogram; (*C*) lung of rat in final stage of poisoning with ANTU.

Thus, in summary, these observations have shown that after ANTU poisoning, fluid appeared in the pleural cavities at the same rate with all dosages above and including the sublethal dose of 3 mg. per kilogram. Dosages many times the LD50 did not increase the rate of the production of the fluid. The fact that all of the rats that received the 3 mg. per kilogram dose would have survived even though they had maximal amounts of fluid in their pleural cavities, demonstrates that the mere presence of very large amounts of fluid in the pleural cavity doses not kill the animals.

### B. PULMONARY EDEMA

From the work of Latta[4] it is known that fluid first becomes visible in the lymph ducts and perivascular spaces near the hilum, next in the walls of the alveoli, later in the alveoli themselves. The microphotographs of lung sections in Fig. 6 show two of the stages of poisoning with ANTU, 50 mg. per kilogram; (*A*) lung of rat killed four hours after poisoning. The interalveolar spaces are filled with fluid and the alveoli contain small amounts of fluid; (*B*) lung of rat killed eighteen hours after poisoning. There are large pools of fluid around the vessels. The lymph ducts are markedly distended with fluid; (*C*) lung of a rat in the final stages of poisoning. Fluid fills most of the alveoli except for a few areas where they are distended with trapped air. The alveoli just under the pleura in Fig. 6 *C* seem to be almost completely filled with fluid. In general, the fluid gathers in the alveoli just under the pleura before it appears in the alveoli in other parts of the lung.

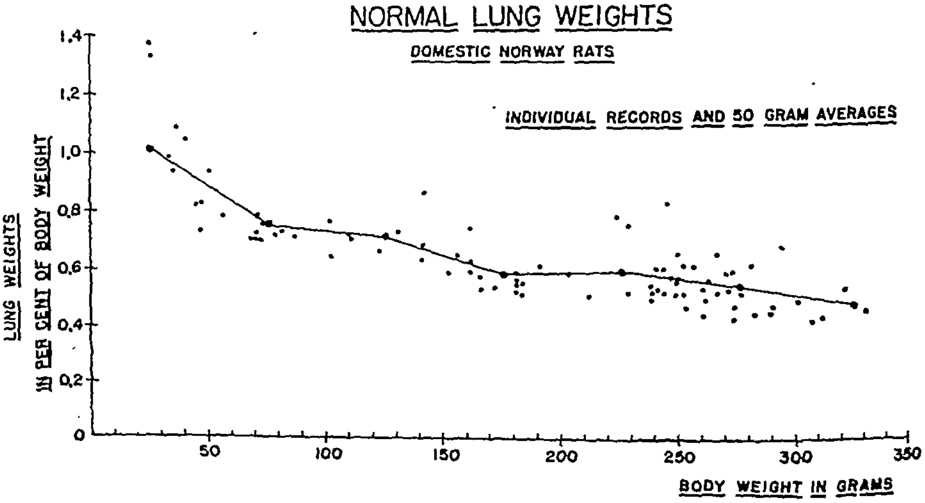


Fig. 7.—Records of lung weights for normal rats of various body weights. Ordinates represent the lung weights in per cent of body weight; the abscissas body weight in grams. Each dot represents the lung weight of an individual rat. The solid line connects the averages for 50-gram intervals.

*Normal Rats.*—

*Lung Weights and Body Weight:* Fig. 7 shows the relation of lung weight to body weight for ninety normal rats. Each point represents one rat. Body weight is given in grams on the abscissas and the lung weight in per cent of body weight on the ordinates. The line passes through the average values for each 50-gram range of weight. The proportionate lung weights decreased at a fairly steady rate from 1.01 per cent, for the rats weighing less than 50 Gm., to 0.46 per cent for the 300 to 350 Gm. group, remaining very nearly constant over a body weight range of 150 to 350 Gm. For the 200 to 250 Gm. rats, the mean was 0.56 per cent of body weight with a standard deviation of 0.02.

*ANTU Poisoned Rats.*—

*Lung Weights:* Fig. 8 shows the lung weights as per cent of body weight with relation to time after injection of the ANTU for the five different doses. The ordinates show the lung weights in per cent of body weight; the abscissas time in hours. The points on the solid curves represent averages for single hour periods while the dotted lines show the average lung weights for normal rats of the same weight range. The amount of edema is thus estimated from the excess of these average lung weights over the normal averages.

*Rate of Increase of Pulmonary Edema:* The rats on the lowest dose, 1 mg. per kilogram, did not show lung edema at any time, while all of the rats on the four higher doses showed a definite edema. During the first hour the average lung weights did not increase in any of the groups of rats; during the second hour, however, they increased in all groups; and during the next two to four hours either continued increasing or reached plateau levels. After that the average lung weights decreased again, the remaining level of edema roughly varying in direct proportion to the dosage. The rats receiving the highest dose still had a marked pulmonary edema at the end of ten hours, shortly before many of them began to die, whereas the rats on the 3 mg. per kilogram dose were practically free of pulmonary edema at this time.

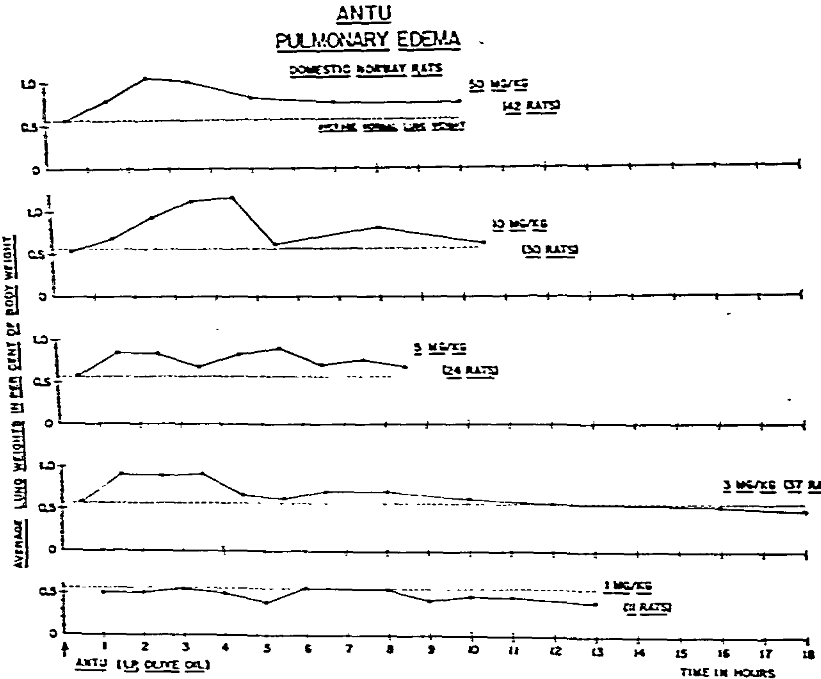


Fig. 8.—Curves showing the average pulmonary edema of rats at hourly intervals after poisoning with five different doses of ANTU. Ordinates represent the lung weights in per cent of body weight; the abscissas time in hours. The dots represent averages for all animals observed within each hour period. The interrupted lines show the average lung weights for normal rats weighing from 200 to 300 Gm. Differences in weight between the lungs after poisoning and the normal average represents the amount of the edema.

The edema amounted to 0.5 per cent of body weight in some instances; in others to only about 0.3 per cent. This means that the weight of the fluid in the lungs at its highest level, was approximately equal to the weight of the lungs.

It should be noted that the changes in lung weights as reported here are based on weighings performed only after the lungs were freed of blood by the technique described for bleeding the rats to death. Since many workers do not follow this procedure, the two records shown in Fig. 9 are presented from our earlier experiments in which we killed the rats by methods other than bleeding. These observations were made on recently trapped wild Norway rats but the results may be compared with those obtained in domesticated rats since they have about the same proportionate lung weights and essentially the same LD50 for ANTU. The dotted horizontals give the average lung weights for normal rats and the bars represent lung weights for the individual experimental rats. In these experiments the rats were given 10 mg. per kilogram of ANTU intraperitoneally in olive oil, and then at various intervals they were killed by crushing the cervical cord. Their lungs were then removed either at once or after thirty to sixty minutes.

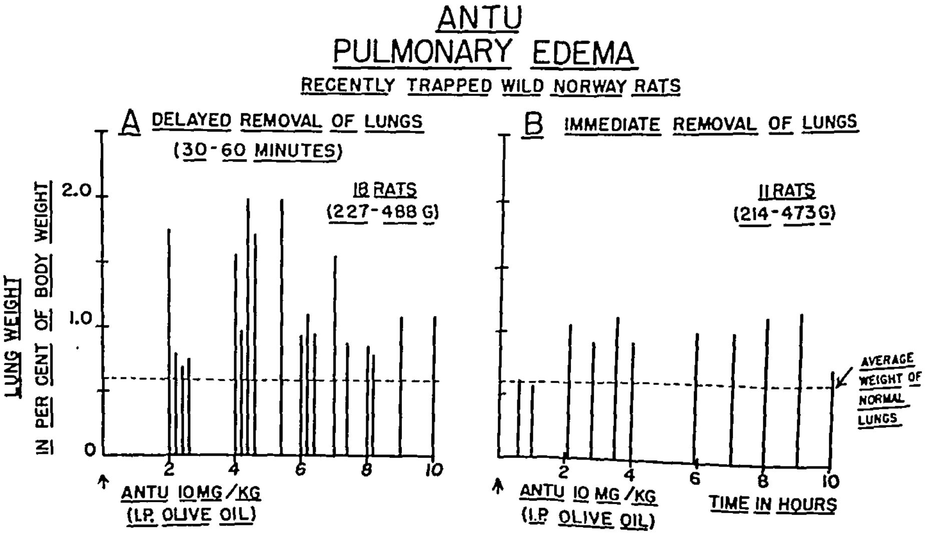


Fig. 9.—Bar graphs showing the lung weights for rats poisoned with ANTU, 10 mg. per kilogram, but not exsanguinated; (*A*) rats that were not autopsied for thirty to sixty minutes after being killed; (*B*) rats that were autopsied as soon as possible after being killed. Each bar represents an individual rat. The dotted lines give the average weight for lungs for normal rats of the same body weight range.

Fig. 9*A* shows that the rats autopsied after a delay had lung weights varying over a very wide range, with differences as much as fourfold. These inconsistent weights probably resulted from the indeterminate amounts of blood pumped into the lungs after the cord was crushed. Fig. 9 *B* shows the greater uniformity in weights of lungs removed as soon as the rats were killed. The increase in lung weights produced by ANTU is clearly demonstrated, but the decrease that occurs after the third and fourth hour in our present experiments was not observed. Presumably the blood in the lungs masked this decrease.

Thus, in summary these observations have shown that after ANTU poisoning, there was marked pulmonary edema for a period lasting from two to six hours. After several hours, depending on the dosage, some or all of the excess fluid left the lungs. The edema was maintained longest in rats that received the largest doses.

It is likely that the fluid that left the lungs was contained in the lymph ducts, perivascular spaces, and alveolar walls, from which it could readily be absorbed through the right and left thoracic ducts or ultimately be aspirated to the pleural cavities. This will be discussed later. On the other hand it is likely that the fluid remaining in the lungs of rats with the higher dosages of ANTU was contained in the alveoli and alveolar ducts from which places, according to the recent results of Courtice and Simmonds,[11] it probably would not be absorbed for several days.

Fluid does not fill many of the alveoli, or the bronchi and trachea until just before death. In some instances, large amounts of fluid leave the trachea after death, literally forming pools around the heads of the dead animals.

### C. THE INTERRELATIONSHIP OF PULMONARY EDEMA AND PLEURAL EFFUSION

After separate consideration of the formation of pulmonary edema and pleural effusion, it is now possible to investigate how they are related—to establish whether fluid appears in the lungs and pleural cavities simultaneously or in sequence. This is significant in order to be able to determine whether the pleural fluid comes from the lungs or whether it comes from the visceral or parietal pleura.

For this purpose, poisoned rats were killed and autopsied at frequent intervals during the first three hours after ANTU injection.

*Onset of Effect:* Fig. 10 exhibits the pleural effusion and lung weight curves for four doses of ANTU (3, 5, 10, and 50 mg. per kilogram) with each point representing the sacrifice of an individual rat. Average lung weights and pleural effusion in normal rats, and their standard deviations, are indicated by dotted lines.

Fig. 10*A* gives the curves for the rats on the 3 mg. per kilogram dose. It shows that the lungs of rats killed at thirty-three and fifty minutes were slightly heavier than the normal average. However, since these weights fall within the standard deviation they can have only a doubtful significance. During the same time, the pleural effusion remained within the average normal levels. By one hour and seven minutes, the lung weights had increased to 1.0 per cent of body weight, which represented almost a maximum increase. In the same period of time the pleural effusion increased to 0.15 per cent, which is only about one-fortieth of the maximum capacity of the pleural cavities. The pleural effusion did not show a marked increase until near the end of the second hour.

Fig. 10*B* gives the record for the rats that received the 5 mg. per kilogram dose of ANTU. Here again the lung weights showed doubtful increases during the first hour, and a near-maximum increase just after the end of the first hour, when the pleural effusion still showed only the slightest increase, if any. The pleural effusion did not show a marked increase until the early part of the third hour.

Figs. 10*C* and *D* give the records for the rats that received the 10 and 50 mg. per kilogram dose, respectively, again showing that the lungs became well-filled with fluid before appreciable amounts of fluid entered the pleural cavities. However, it should be noted that the increase in both lung weight and pleural effusion occurred more gradually on the higher doses.

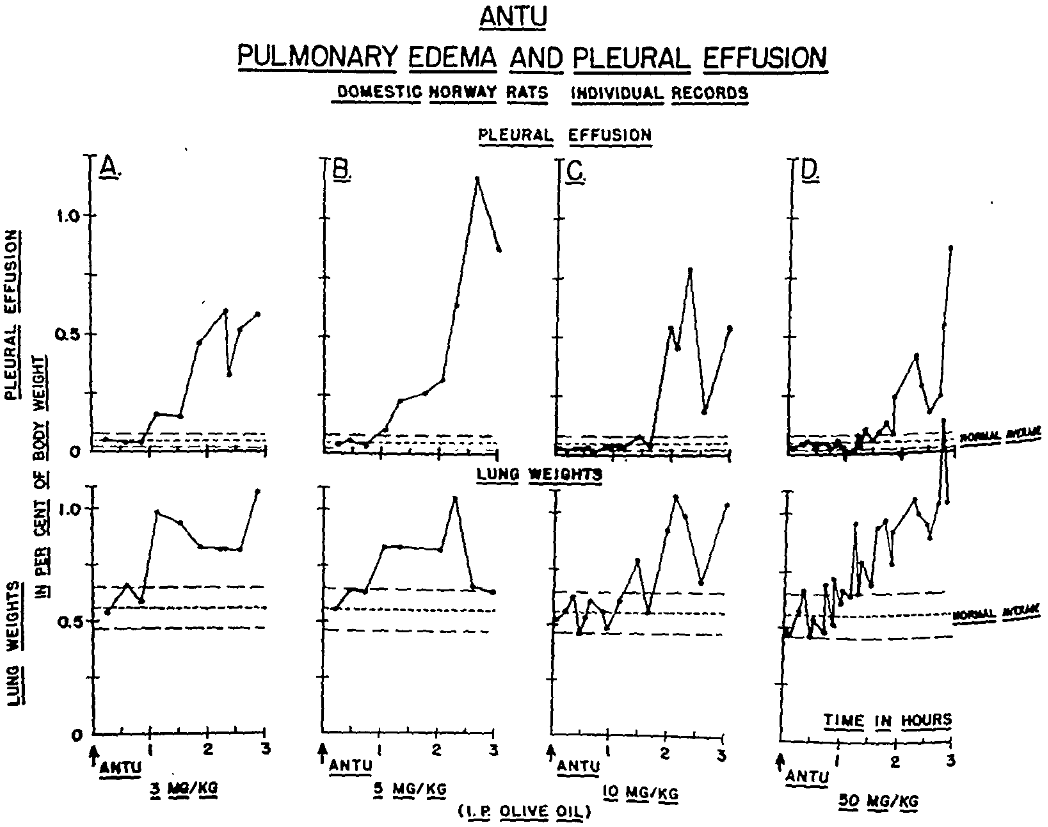


Fig. 10.—Curves showing the temporal relationship between the appearance of pulmonary edema and pleural effusion after various doses of ANTU. Each pair of dots represents data from a single rat. The interrupted lines give the average level and its standard deviation for normal rats.

A study of the curves of Fig. 5 shows that for rats on the 3 mg. per kilogram dose, the pulmonary edema began to recede during the fourth hour, at the time that the pleural effusion was increasing at a very rapid rate. The records for the rats on the other doses show essentially the same relationship.

In summary these observations indicate that after ANTU poisoning, pulmonary edema develops before any appreciable amount of fluid appears in the pleural cavity, and conversely, that the pulmonary edema begins to recede about the time that large amounts of fluid enter the pleural cavity.

These observations indicate that fluid does not appear in the pleural cavities until the lungs have become well filled. We may conclude that the pleural fluid probably comes from the lungs rather than from the visceral or parietal pleurae. In other words the drug affects the permeability of the capillaries of the lungs only, not of those in the pleura or any other parts of the body. Drinker[5] has pointed out that the lung capillaries are particularly assailable also by anoxia, the chief reason being that these capillaries get their oxygen directly from the air that comes into contact with them, whereas the capillaries of other parts of the body get their oxygen from the common pool of arterial blood supply.

On the basis of clinical and experimental observations, Graham concluded in 1921 that the pleural fluid comes from the lungs.[12] He showed that respiration plays the most important part in the shift of fluid from the lungs to the pleural cavity. According to his thesis, the negative pressure in the thorax of an individual with edematous lungs suffices during the inspiratory phase to aspirate fluid from the lungs to the pleural cavity. The deeper the inspirations the more fluid finally gets aspirated to the pleural cavity. The fluid does not actually leave the lungs during the inspiratory phase, but is brought close to the pleural surface, from where during the following expiration, through shrinking of the lungs, the fluid is pressed out into the pleural cavity.

Graham does not mention through what channels he believes the fluid flows from the lungs to the pleural cavity. Within the lungs it is likely that much of the fluid is conducted to the pleurae through the lymph ducts. Maximow and Bloom state that when for any reason the lymph ducts that lead to the hilum nodes become blocked, fluid flows through the lymph ducts that follow the veins to the pleurae.[13] They state also that valves in the lymph ducts prevent the fluid from flowing back into the lungs.

Early after poisoning with ANTU the rats show changes in breathing, first a mild dyspnea, later heavy deep labored inspirations, strictly of the thoracic type.

Thus, if we apply to the ANTU poisoned rats, the line of reasoning used by Graham, it would seem likely that progressively as the fluid appears in the lungs, or as the air spaces around the capillaries become decreased, breathing becomes deeper. Consequently, with each inspiration fluid is aspirated through the pleura and out into the pleural cavity. Once the fluid gets into the pleural cavity it cannot return to the lungs. The more the lungs become compressed, the smaller the breathing areas become, and as a result the more exaggerated the inspiratory efforts become. In this process all the excess fluid that can leave the lungs becomes aspirated into the pleural cavity. This may explain the decline of the edema that was reported above. It is unlikely that fluid that has already crossed to the alveolar spaces will be forced back into the lungs. The amounts of fluid that get into the alveoli probably determines the fate of the rats.

### D. BLOOD VOLUME

*Normal Rats.—*

*Blood Weight and Body Weight:* The curve in Fig. 11 shows the amount of blood that was obtained from eighty-six normal rats of different body weights.

Ordinates represent the blood weight in per cent of body weight, the abscissas body weight in grams. Individual points on the curve represent averages for body weight ranges of 50 Gm. It will be seen that the blood weight is essentially constant in relation to body weight for rats between 100 and 250 Gm. As in the case of the lungs, the relative weight of these essential tissues is greater in the younger (small) animals, and distinctly less in animals weighing over 300 Gm. (with their greater proportion of body fat). For the 200 to 299 Gm. rats the mean was 3.5 per cent of body weight, with a standard deviation of 0.37, while in rats up to 50 Gm. the blood represented an average of 4.6 per cent of body weight.

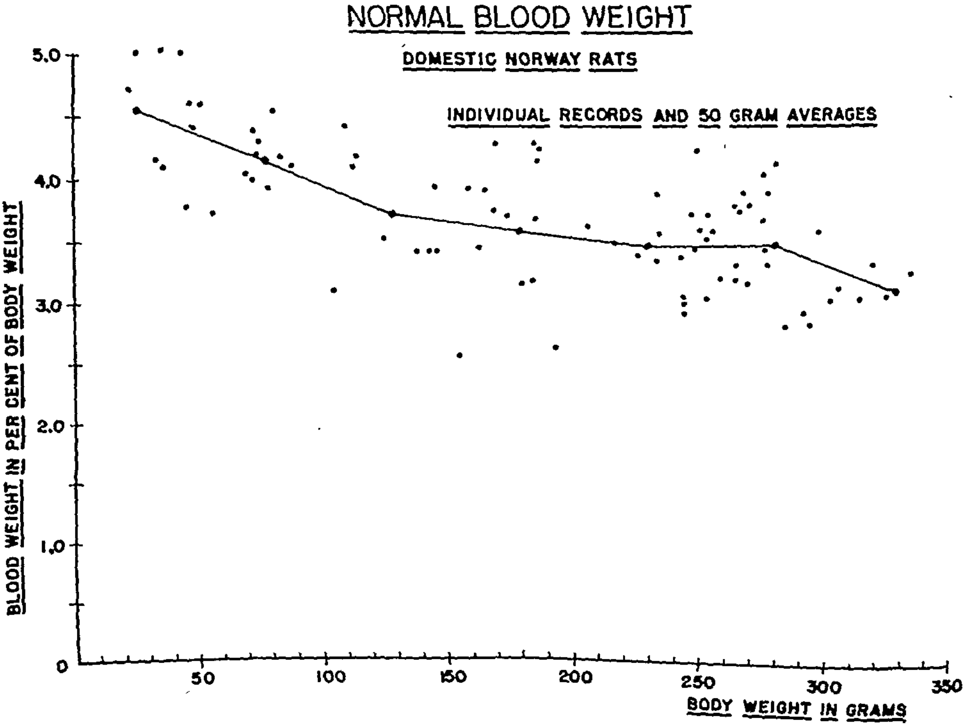


Fig. 11.—Chart showing the amounts of blood obtained by bleeding normal rats to death, (blood swabbed after cutting vena cava and abdominal aorta). Each dot represents the blood weight for one rat. The solid line connects the average blood weights for each 50-gram interval.

These weights were determined by the method of swabbing with cotton as described previously. They fall short of determinations made by other workers, presumably because the method does not assure complete withdrawal of the blood. Some workers have reported a total blood weight of 4 per cent; however most of them give values near 6 per cent.[14]

*ANTU Poisoned Rats.*—

*Blood Weights:* Fig. 12 shows the blood weight curves for the rats poisoned with the five different dosages of ANTU. Ordinates give the blood weights in per cent of body weight; abscissas time in hours after the injection of ANTU. The points on the curves represent averages of each hour up to nine hours; after that averages of two- to three-hour periods. As might have been anticipated from the other data, the blood weights of the rats on the 1 mg. per kilogram dose remained entirely unaffected by the drug. The blood weights of the rats on the 3 mg. per kilogram dose decreased from 3.6 per cent to 2.5 per cent in the course of five hours, thereafter rising to more than 3 per cent. On the three higher doses, the blood weights decreased from levels near 3.5 per cent to a level near 1.0 per cent in seven to ten hours. A comparison of all four curves with those for the pleural effusions in Fig. 4 and Fig. 8 shows that the blood weights decreased roughly in proportion to the increases in pleural effusion in the first five hours.

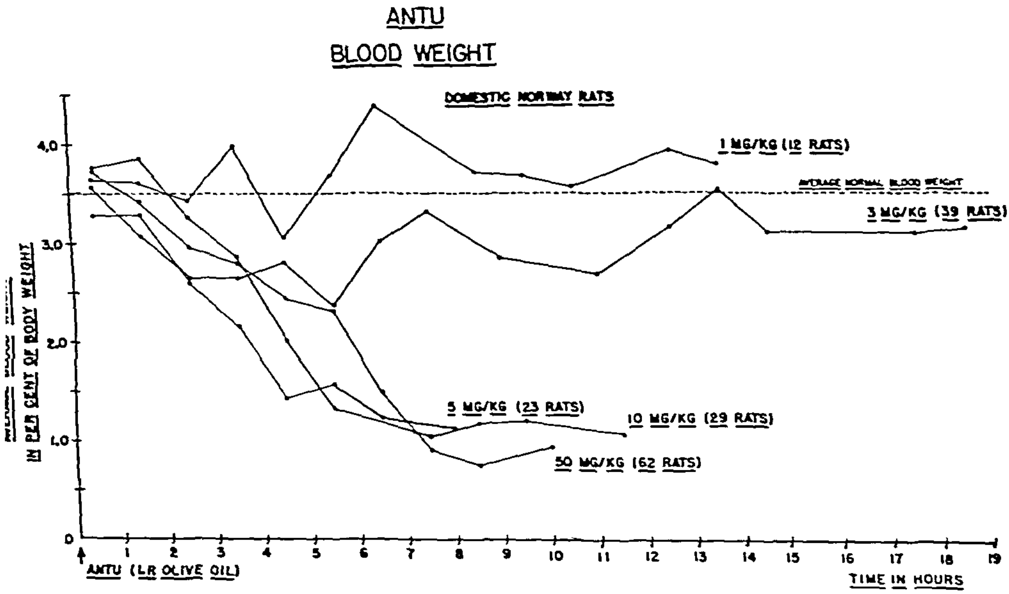
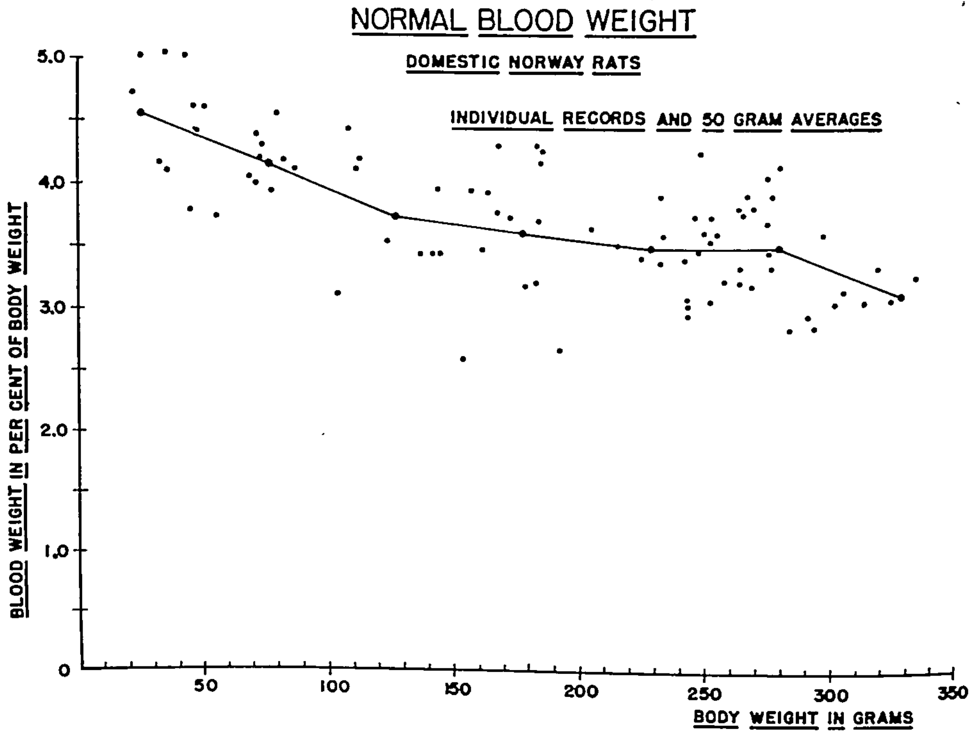


Fig. 12.—Curves showing the average amount of blood obtained by bleeding ANTU poisoned rats to death. Ordinates represent the blood weight in per cent of body weight; the abscissas time in hours. Each dot represents the average for rats observed during each hour after poisoning. The curves are given for five doses of ANTU.

At the time when the blood had been reduced to 1 per cent of the body weight it had a dark color and a viscous consistency. Again as might be anticipated, hematocrit determinations (Wintrobe) showed that the blood began to become concentrated at about the same time as fluid first appeared in the pleural cavities and that thereafter the concentration and pleural effusion increased at much the same rate, while there was a corresponding decrease in blood weight. Fig. 13 exhibits hematocrit values of the rats on the 5 mg. per kilogram dose of ANTU. Fig. 14 shows the hematocrit values of the rats that received the sublethal dose of 3 mg. per kilogram.

Ordinates represent the blood weight in per cent of body weight, the abscissas body weight in grams. Individual points on the curve represent averages for body weight ranges of 50 Gm. It will be seen that the blood weight is essentially constant in relation to body weight for rats between 100 and 250 Gm. As in the case of the lungs, the relative weight of these essential tissues is greater in the younger (small) animals, and distinctly less in animals weighing over 300 Gm. (with their greater proportion of body fat). For the 200 to 299 Gm. rats the mean was 3.5 per cent of body weight, with a standard deviation of 0.37, while in rats up to 50 Gm. the blood represented an average of 4.6 per cent of body weight.



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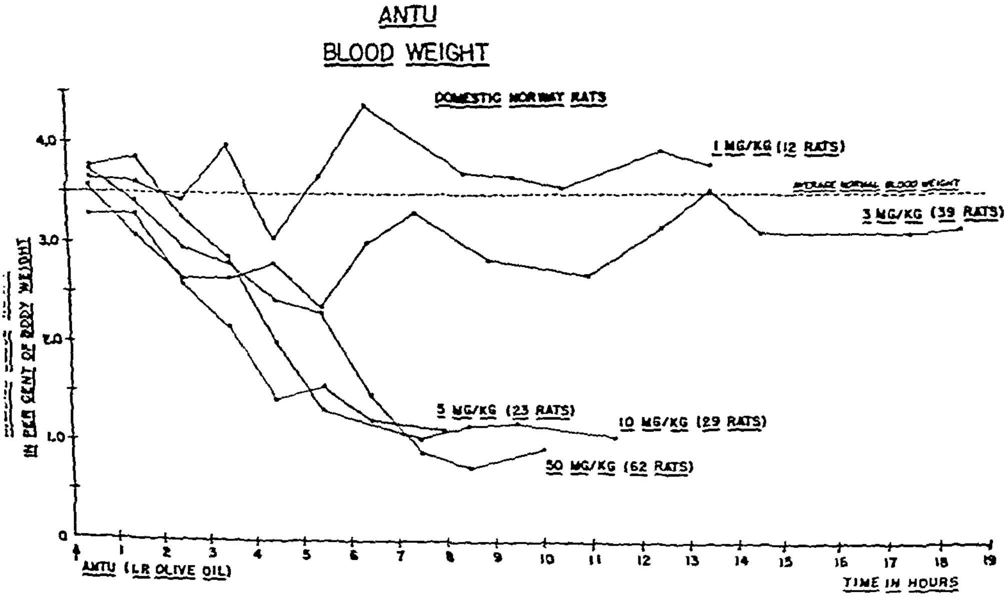

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At the time when the blood had been reduced to 1 per cent of the body weight it had a dark color and a viscous consistency. Again as might be anticipated, hematocrit determinations (Wintrobe) showed that the blood began to become *concentrated at about the* same time as fluid first appeared in the pleural cavities and that thereafter the concentration and pleural effusion increased at much the same rate, while there was a corresponding decrease in blood weight. Fig. 13 exhibits hematocrit values of the rats on the 5 mg. per kilogram dose of ANTU. Fig. 14 shows the hematocrit values of the rats that received the sublethal dose of 3 mg. per kilogram.

Thus, in summary, these observations show that after ANTU poisoning, the large amounts of fluid that fill the lungs and pleural cavities have come to a large extent from the extracellular fluid of the blood. However, in instances in which the fluid in the lungs and pleural cavities amounts to 4 to 6 per cent of the body weight, the approximate equivalent of the total blood volume, it is clear that some of the fluid must also come from the interstitial sources of the extracellular supply.

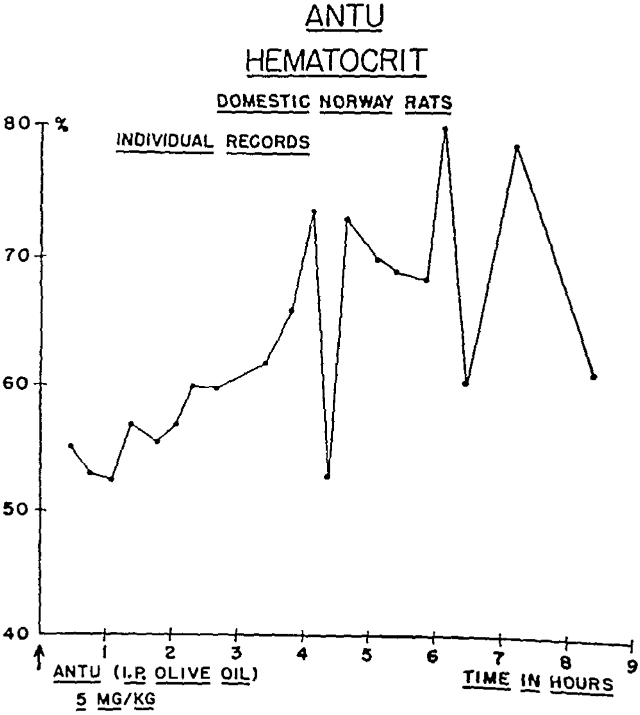

Fig. 13.—Curves showing the hematocrit readings for individual rats at various intervals after receiving 5 mg. per kilogram of ANTU by intraperitoneal injection.

## CHEMISTRY AND CYTOLOGY OF THE BLOOD AND PLEURAL EFFUSION OF ANTU POISONED RATS

Chemical and cytologic studies of the blood and pleural effusion were not made at regular intervals after poisoning with ANTU. This will be done in a later study. Observations were made only on a small number of animals that were killed and autopsied about six hours after having received a 50 mg. per kilogram dose of ANTU. However, the incomplete data thus obtained may help to throw some further light on the characteristics of the pleural effusion and blood of ANTU poisoned rats and of their interrelationships.

Table I shows that the content of sodium and potassium in the pleural fluid of ANTU poisoned rats is essentially the same as in the blood serum of normal rats.

Table II shows that the protein and total nitrogen content of the pleural effusion was high, in fact about as high as that of the blood plasma, either of normal or of ANTU poisoned rats.

Table III shows that the white blood cell count of control normal rats averaged 13,630, which is higher than the counts of 11,556 reported by Scarborough,[15] and 11,931 by Wills and Mehta,[16] but lower than the count of 18,819 reported by Thewlis and Meyer.[17] In the ANTU poisoned rats the count increased to 17,455, which may represent a significant increase over the average for normal rats. The count for the pleural effusion was very much lower, 1,770; in some rats it was as low as 700.

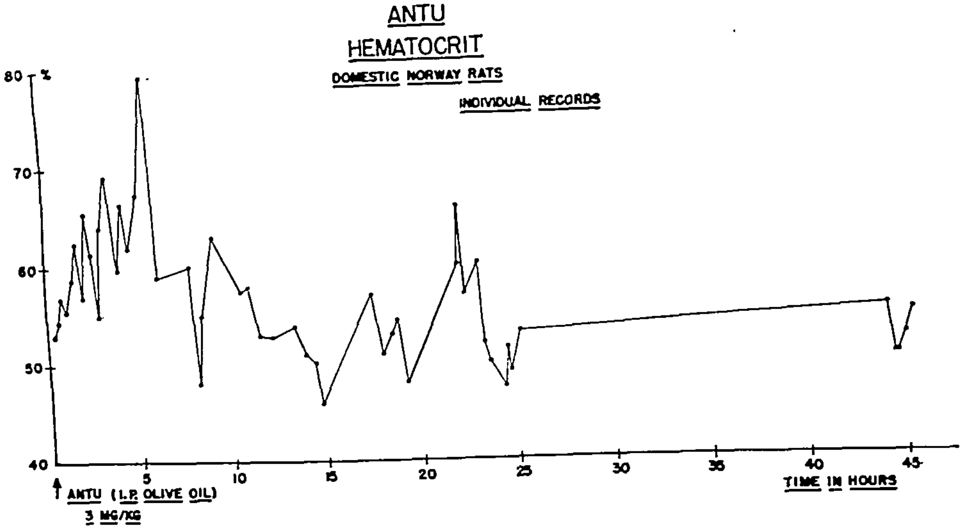

Fig. 14.—Hematocrit readings for individual rats in various intervals after receiving 3 mg. per kilogram of ANTU, by intraperitoneal injection.

TABLE I. AVERAGE SODIUM AND POTASSIUM CONTENT OF BLOOD SERUM AND PLEURAL EFFUSION

| | SODIUM MEQ/1 | POTASSIUM MEQ/1 |
|---|---|---|
| Normal Rats (8) | 144.1 | 4.6 |
| Blood serum | (139.6-148.3) | (3.7-4.8) |
| ANTU-poisoned rats (5) | 136.0 | 4.1 |
| Pleural fluid | (129.0-145.0) | (3.4-4.3) |

TABLE II. AVERAGE SPECIFIC GRAVITY AND PROTEIN CONTENT OF BLOOD PLASMA AND PLEURAL EFFUSION

| | SPECIFIC GRAVITY | PROTEIN GRAMS PER CENT | TOTAL NITROGEN GRAMS PER CENT |
|---|---|---|---|
| Normal Rats (8) | 1.024 | 5.91 | 1.042 |
| Blood Plasma | (1.023-1.026) | (5.5-6.5) | (0.926-1.168) |
| ANTU-Poisoned Rats (8) | 1.024 | 5.81 | -- |
| Blood Plasma | (1.021-1.028) | (4.8-7.2) | |
| Pleural Fluid | 1.020 | 4.74 | 0.675 |
| | (1.018-1.022) | (3.8-5.2) | (0.604-0.734) |

TABLE III. AVERAGE CELL CONTENT FOR BLOOD AND PLEURAL EFFUSION

| | W.B.C. | MONO-CYTES | LYMPHO-CYTES | NEUTRO-PHILS | EOSINO-PHILS | JUVE-NILES |
|---|---|---|---|---|---|---|
| Normal Rats (11) | 13,630 | 0 | 70.4 | 25.7 | 3.0 | 0 |
| Blood | (5,650-29,250) | - | (44-83) | (14-49) | (1-7 ) | - |
| ANTU-Poisoned Rats | 17,455 | 0 | 50.0 | 47.0 | 1.0 | 2.0 |
| Blood (18) | (6,050-29,550) | (0-1) | (38-56) | (43-58) | (0-1 ) | (0-5) |
| Pleural Effusion | 1,770 | 2.3 | 76.4 | 3.5 | 14.8 | 3.0 |
| (15) | ( 700- 3,925) | (0-10) | (48-96) | ( 0-14) | (3-40) | (0-8) |

The next five columns give the differential count of the white blood cells. In the blood of normal rats the percentages of lymphocytes were almost three times as high as that of the neutrophils and there were only a few eosinophils. In the blood of ANTU poisoned rats the percentage of lymphocytes was reduced, and correspondingly the percentage of neutrophils was increased.

The pleural effusion contained a high proportion of lymphocytes, almost no neutrophils, and many eosinophils. These results show that lymphocytes and eosinophils pass into the pleural cavity very readily, but neutrophils pass only in very small numbers if at all.

### BEHAVIOR CHANGES

During the first thirty to sixty minutes after poisoning the animals tended to become less active. Thereafter they sat curled up in a corner of their cage and neither ate nor drank, until they were sacrificed or died, that is providing they received a lethal dose. When they received a sublethal dose they started to eat and drink, and to become active at varying times after the eighth to tenth hours.

### OTHER PHYSIOLOGICAL CHANGES

At the same time when fluid began to fill the lungs, body temperature dropped sharply,[2] gastric evacuation was delayed,[18] and the rats became anuric.[19]

### GENERAL SUMMARY OF DISPOSITION OF EXTRACELLULAR FLUID AFTER ANTU POISONING

On the basis of the results of the present studies, and of the work of Drinker,[5] Drinker and Hardenbergh,[20] Latta,[4] Graham,[12] an attempt may now be made to give a schedule of the events involved in the disposition of the extracellular fluid after it leaves the lung capillaries of ANTU poisoned animals.

1. The fluid surrounds the capillaries and flows through the interalveolar spaces until it reaches the lymph ducts and is absorbed.

2. In the lymphatic ducts it is carried toward the hilar nodes and from there to the right and left thoracic lymph ducts that lead to the subclavian veins.

3. The fluid very soon appears in large amounts in the perivascular spaces, forming pools of fluid that surround not only the blood vessels but the lymphatic ducts. How the fluid gets from the lymphatic ducts to these spaces is not known. At the hilum these fluid-filled spaces are readily visible through the pleura.

4. Extracellular fluid leaves the capillaries faster than it can be removed by the right and left thoracic lymph ducts. As a result the fluid is forced to flow either into the alveoli or in the opposite direction in the lymph ducts to the pleura.

5. Areas of contact between the air and the lung capillaries are greatly reduced, partly by fluid that surrounds the capillaries, partly by fluid that is present in the alveoli. Both ventilation and gas exchange becomes impaired, and breathing becomes deeper.

6. The deeper inspirations increase the negative intrathoracic pressure and fluid in the lungs near the pleura is aspirated toward the surface. Valves in the lymph ducts prevent the fluid from flowing back into the lungs; so as the lungs contract with each expiration, fluid is squeezed out into the pleural cavity. However, the anatomy of the transudation process by which fluid gets from lymphatics of the visceral pleura to the pleural cavities remains unknown.

7. The pulmonary edema soon reaches a maximum level—fluid fills the interalveolar spaces, the lymph ducts, perivascular spaces, and many of the alveoli, especially those located near the pleura.

8. The fluid that leaves the lungs with each expiration begins gradually to fill the pleural cavities, surrounding and compressing the lungs, and limiting the space available to them.

9. Owing to the compression of the lungs, and the difficulty of achieving a full inflation, the inspirations progressively become deeper and deeper. Fluid is aspirated at such a rapid rate that the lung edema recedes—that is, *less fluid remains in the lymph ducts* and perivascular spaces. Fluid that has already entered the alveoli probably stays there since it cannot readily be absorbed.

10. At a considerably later stage fluid fills the entire pleural cavity, pushing the diaphragm to its limit, well down into the abdominal cavity, and distending the thorax. Fluid that is still escaping from the capillaries, enters the alveoli, and soon asphyxiates the animal. In the last stage fluid fills the bronchi, trachea, and actually flows from the nose to form a pool around the dead animal.

## DISCUSSION

In these studies on rats it was shown how, with the use of ANTU and quantitative methods for the measurement of pulmonary edema and pleural effusion, the course of the flow of extracellular fluid can be followed from the time the fluid leaves the lung capillaries until it becomes distributed to the many parts of the lung, pleural cavities, or returned to the blood stream. It is of special importance that this flow can be thus followed in animals without the complexities that arise in man from presence of infection and defective cardiac function.

On the basis of the results displayed in the charts it should be possible to test various agents, such as morphine, *atropine*, and various surgical procedures, such as sympathectomies, that are reported to have an effect on pulmonary edema and pleural effusion. It should also be possible to test various theories about the origin of these phenomena.

The quantitative methods described in this report may also be used for the study of the pulmonary edema and pleural effusion produced by other agents and surgical interferences, such for instance as vagotomies, brain lesions, nephrectomies, exercise, and stress.

Many of the investigations on pulmonary edema and pleural effusion will have to be done on the rat, since it is fairly expendable. Some investigations can be made, however, on the dog, an animal that, like the rat, gives quite constant and reproducible results. Owing to its large size the dog will in some instances lend itself for experiments for which the rat is too small.

A study of thiourea derivatives[21] other than ANTU may throw further light on the underlying mechanisms.

The extent to which results obtained from the rats and dogs can be translated to man is not known, however, it is likely that the mechanisms involved will not be very different. One of the chief anatomic differences is that the rat and dog have a "thin" pleura, whereas man has a "thick" pleura.[22]

It is well known that pulmonary edema and pleural effusion may appear in man quite as quickly as in the rat. Graham[12] mentioned that in patients suffering from a streptococcal pneumonia as much as 1000 c.c. of fluid had been removed from one thoracic cavity within a day or two after the start of the illness.

Observations made by Yamada and co-workers[23] on the pleural fluid of normal healthy soldiers while resting or after exercising may be pertinent at this point. By thoracentesis Yamada was able to draw out varying amounts of fluid (up to 20 Gm.) from as many as 50 per cent of unexercised soldiers; and larger amounts in 70 per cent of exercised soldiers. It is possible, therefore, that fluid appears far more often in the pleural cavities than might have been suspected.

The prominence of the part played by the perivascular spaces in the lung edema produced by ANTU calls attention to the great lack of knowledge about these structures. That little should be known about them probably is not too surprising, since in the normal lung they are not visible. To what extent they resemble perivascular spaces found in other organs, for instance the brain or liver, we have not been able to decide.

The demonstration of large numbers of eosinophils in the pleural fluid also requires special comment. Clinically eosinophilia of pleural fluid has been found in cases of tuberculosis and lobar pneumonia as well as in hypersensitivity diseases, asthma, and Löeffler's syndrome.[24] The observations reported here show that such eosinophilia may appear in the absence of such states. It is noteworthy that an eosinophilia of pleural effusion and a reduction of eosinophils in the blood stream has been produced with some regularity in patients by pneumothorax.[25, 26]

The ability to produce eosinophilia with a fair degree of constancy with ANTU offers an opportunity to study the appearance of eosinophils in the pleural fluid under experimental conditions, free from complications and infections.

Attention may be drawn to the fact that much larger amounts of fluid can be obtained in ANTU poisoned rats than from the blood of normal rats. The ready availability of such large amounts of plasma may be made use of for a number of different investigations.

The exsanguinating technique used in these experiments was adopted at the suggestion of Dr. Arnold Rich, and the results obtained agree with those reported by Durlacher, Banfield, and Bergner on post-mortem edema.[27] They found that when the lungs of rabbits were weighed immediately after death the method of killing did not make much difference in the observed weights; except that the lungs were lightest when the rats were killed with ether. The microscopic examination showed that the alveolar walls were devoid of blood in animals sacrificed by exsanguination or ether. Animals killed with nembutal, magnesium sulphate, or a blow on the head showed great differences in lung weights when they were not autopsied for several hours; animals killed by exsanguination did not show these changes.

Finally, attention should be drawn to one aspect of the effect of ANTU which has not been mentioned: that it produces all the characteristics of shock. That it should show these characteristics is not surprising when we consider the loss of the great amounts of extracellular fluid that the reaction involves. Along with the increase in hematocrit readings, body temperature drops sharply to very low levels, blood sugar increases, anuria sets in, gastric evacuation is retarded, and the animals stop eating and drinking. These clinical aspects will be discussed in detail in a later paper.

## SUMMARY

1. Quantitative methods are described for the study of the physiology of pulmonary edema and pleural effusion as produced in the Norway rat by alpha-naphthyl thiourea (ANTU).

2. The amount of moisture in the pleural cavities of normal rats weighing 200 to 300 Gm. averaged 0.05 per cent of body weight; the lung weights averaged 0.56 per cent of body weight.

3. The rats were injected with ANTU at five different doses, 1, 3, 5, 10, and 50 mg. per kilogram of body weight and were killed and autopsied at regular intervals. The first two doses are sublethal, the LD50 being 4.1 mg. per kilogram.

4. In some instances pulmonary edema appeared to begin as soon as thirty minutes after ANTU injection; in most animals it was well started by the end of the first hour. It reached a maximum, 0.5 to 0.8 per cent of body weight, during the second and fourth hours and thereafter either decreased or resulted in death, depending on the dosage.

5. The pleural effusion began after the first hour and increased sharply during the second hour. For the next six to ten hours it increased at a constant

and rapid rate, reaching levels near 3.5 per cent. In some animals it amounted to 5 to 6 per cent.

6. The rate of production of pleural fluid present during the first ten hours was essentially independent of the dosage of ANTU with doses above 3 mg. per kilogram.

7. In rats that received sublethal doses of ANTU the fluid in the lungs and pleural cavities was quickly absorbed within fifteen to twenty hours or less in some instances.

8. The lungs definitely became well filled with fluid before any appreciable amounts of fluid appeared in the pleural cavities.

9. The amount of blood recovered from bleeding normal rats to death averaged 3.6 per cent of body weight for rats weighing from 200 to 299 Gm.

10. After ANTU poisoning these amounts of recovered blood decreased to levels as low as 1.0 per cent, when the blood was very thick and dark in color.

11. The hematocrit increased to levels as high as 80 within ten hours after poisoning.

12. Chemical studies showed that the pleural effusion and blood contained the same amounts of sodium and potassium.

13. The protein content of the pleural effusion averaged 4.44 Gm. per cent (normal for blood 5.91); total nitrogen 0.675 Gm. per cent (normal 1.042).

14. Cytological studies showed that the total number of white blood cells in the blood plasma was somewhat higher in ANTU poisoned rats than in normals. The pleural effusion contained only about one-tenth as many white blood cells as were present in the blood serum of the ANTU poisoned rats. The plasma of normal rats contained about three times as many lymphocytes as neutrophils and only a few eosinophils, whereas in the plasma of ANTU poisoned rats the lymphocytes and neutrophils were present in almost equal numbers, and eosinophils were fewer, if anything. The pleural effusion contained almost no neutrophils, and a correspondingly higher percentage of lymphocytes. It also contained a much higher percentage of eosinophils.

15. The various steps probably involved in the production and control of the pulmonary edema and pleural effusion were discussed.

16. It was pointed out that the curves for the pulmonary edema and pleural effusion may also be used as base lines for testing the effects of various agents, such as vagotomy, nephrectomy, exercise, and stress.

These experiments were carried out with the help of a number of workers who assisted me at various times after the start of the investigation in 1943. Dr. Sally H. Dieke, Mrs. Kathryn H. Clisby, Dr. George S. Allen, Mr. Henry N. Wagner, Jr., Dr. Katherine K. Rice, and Mr. John O. Neustadt helped in the preparation of the manuscript, and Mr. Thomas A. Stebbins and Zolton Yuhasz made the drawings. I am also indebted to Drs. Cecil K. Drinker, Arnold R. Rich, Henry M. Thomas, Jr., Richard Riley and Donald F. Proctor for helpful suggestions and criticisms.

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and rapid rate, reaching levels near 3.5 per cent. In some animals it amounted to 5 to 6 per cent.

6. The rate of production of pleural fluid present during the first ten hours was essentially independent of the dosage of ANTU with doses above 3 mg. per kilogram.

7. In rats that received sublethal doses of ANTU the fluid in the lungs and pleural cavities was quickly absorbed within fifteen to twenty hours or less in some instances.

8. The lungs definitely became well filled with fluid before any appreciable amounts of fluid appeared in the pleural cavities.

9. The amount of blood recovered from bleeding normal rats to death averaged 3.6 per cent of body weight for rats weighing from 200 to 299 Gm.

10. After ANTU poisoning these amounts of recovered blood decreased to levels as low as 1.0 per cent, when the blood was very thick and dark in color.

11. The hematocrit increased to levels as high as 80 within ten hours after poisoning.

12. Chemical studies showed that the pleural effusion and blood contained the same amounts of sodium and potassium.

13. The protein content of the pleural effusion averaged 4.44 Gm. per cent (normal for blood 5.91); total nitrogen 0.675 Gm. per cent (normal 1.042).

14. Cytological studies showed that the total number of white blood cells in the blood plasma was somewhat higher in ANTU poisoned rats than in normals. The pleural effusion contained only about one-tenth as many white blood cells as were present in the blood serum of the ANTU poisoned rats. The plasma of normal rats contained about three times as many lymphocytes as neutrophils and only a few eosinophils, whereas in the plasma of ANTU poisoned rats the lymphocytes and neutrophils were present in almost equal numbers, and eosinophils were fewer, if anything. The pleural effusion contained almost no neutrophils, and a correspondingly higher percentage of lymphocytes. It also contained a much higher percentage of eosinophils.

15. The various steps probably involved in the production and control of the pulmonary edema and pleural effusion were discussed.

16. It was pointed out that the curves for the pulmonary edema and pleural effusion may also be used as base lines for testing the effects of various agents, such as vagotomy, nephrectomy, exercise, and stress.

These experiments were carried out with the help of a number of workers who assisted me at various times after the start of the investigation in 1943. Dr. Sally H. Dieke, Mrs. Kathryn H. Clisby, Dr. George S. Allen, Mr. Henry N. Wagner, Jr., Dr. Katherine K. Rice, and Mr. John O. Neustadt helped in the preparation of the manuscript, and Mr. Thomas A. Stebbins and Zolton Yuhasz made the drawings. I am also indebted to Drs. Cecil K. Drinker, Arnold R. Rich, Henry M. Thomas, Jr., Richard Riley and Donald F. Proctor for helpful suggestions and criticisms.

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One hundred eighteen, or 51.3 per cent, of those living in Groups I and II have been followed from five to seventeen years and have arrested or apparently cured tuberculosis, whereas 13, or 5.6 per cent, of this group still have active disease. Of those living and followed less than five years, 64, or 27.8 per cent, have arrested or apparently cured disease. In combining the two groups we find that 194, or 84.3 per cent, of those living or dying from causes unrelated to tuberculosis had or have arrested or apparently cured tuberculosis. Thirteen, or 5.7 per cent, are apparently well but have positive sputum or gastric cultures, and 35, or 15.2 per cent, have active tuberculosis.

## COMMENT

Although the number is small and not statistically significant, there were no striking differences noted in results in the various age, sex, or racial groups. Better results in the "good chronic" have generally been observed, and hold true in this series, although nine of ten patients with caseous pneumonic disease have sputum conversion on culture, and one is positive on culture only. All patients had one or more courses of streptomycin prior to and during operation. Poorer results have been observed in giant cavitary lesions, in markedly destructive disease, and where there is tuberculous tracheobronchitis. Similar observations were made by Rubin and Klopstock.[2] Nineteen patients in the series had concomitant systemic diseases, such as diabetes, cardiovascular disease, peptic ulcer, and hyperthyroidism. Twenty-nine had respiratory complications such as asthma, bronchitis, bronchiectasis, pneumonoconiosis, and emphysema. With the use of sulfonamides and penicillin in this series, secondary wound and pulmonary infections were seldom encountered. Streptomycin and other antituberculosis drugs were often used in recent years during the operative period to prevent or reduce postoperative spread. Physiotherapy, including preoperative and postoperative exercises, has been used in recent years with noticeably less chest deformity.

## LATE RESULTS REPORTED IN THE LITERATURE

Results in the literature during the last two decades[1, 3-11] reveal improvement in the majority of patients operated upon, although consistency in evaluating results of sputum conversion makes comparison difficult. Reports on sputum conversion, the best evidence of a lasting and permanent result, are often omitted, and, if given, some reports of conversion are based on microscopic absence of the tubercle bacillus on direct smear or on concentrate only. In recent years an increasing number includes the more dependable cultural studies of sputum or gastric washings, or both, and animal inoculation. In the original series, 63 per cent of patients converted on repeated examination of concentrated sputum. Animal inoculation was not routinely done on all. The late results in this series have included sputum or gastric culture in most instances. In the second series of 188, all results are based on conversion of sputum or gastric washings on culture.

In evaluating the late results in thoracoplastry for pulmonary tuberculosis, Dolley, Jones, and Paxton,[7] in 1939, found a total of 1,636 cases reported in the literature, and 43 per cent were improved. In 1941 Skinner and associates[12] reported that of 100 cases followed from 1924 to 1941, 54 per cent were "apparently cured." Douglass[13] in 1947 reported that 69 of 102 patients subjected to thoracoplasty were classified as "good," which requires that sputum be absent or microscopically negative at the fifth year after operation. Of 168 patients reported by Rubin and Klopstock[2] in 1949, 141 were followed for a period of from two to six years, and of these 63.1 per cent had arrested disease with negative sputum or gastric concentrates on culture. Kinsella and coworkers[14] in 1949 reported that of 613 patients receiving thoracoplasty for pulmonary tuberculosis at Glen Lake Sanatorium, followed from five to twenty-six years, 66.8 per cent were alive, and 88.1 per cent of patients with positive sputum converted to negative "according to the direct examination technique used at the sanatorium over the years covered by the study." Recently, Gutheil, Steele, Cadden, and Sakaguchi[15] reported results of thoracoplasty for pulmonary tuberculosis in 254 patients, followed for two to ten years. Cultures of pulmonary secretions were negative in 193, 67 per cent of patients. Hagn-Meincke[16] reported late results in 420 cases subjected to thoracoplasty at the Oresunds-hopsital, Copenhagen, Denmark, followed for less than one-half to more than two years. At the end of the observation period, 193, 46 per cent, were classified as "healthy," which included negative cultures of sputum or gastric washings. Follow-up is not sufficiently long in this series to evaluate late results.

## SUMMARY AND CONCLUSIONS

From 1932 through July, 1948, 334 patients received thoracoplasty for pulmonary tuberculosis at Grasslands Hospital. Follow-up through 1949 (one to seventeen years) reveals that 230 are living and 104 are dead from all causes. One hundred ninety-four, or 58.0 per cent, attained arrest or apparent cure of the disease, 12 of these died from unrelated causes; 13, 3.8 per cent, are symptom free and positive only on culture of sputum or gastric washings; and 35 have active disease. Results reported here compare favorably with other reports on late results from thoracoplasty and emphasize the competence of thoracoplasty in the treatment of pulmonary tuberculosis. Among the 230 living patients, and including 12 who died from causes unrelated to tuberculosis after arrest of their disease, 84.3 per cent had or have arrested or apparently cured tuberculosis. Late results in thoracoplasty justify its prominent place in the surgical treatment of pulmonary tuberculosis.

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# THE SURGICAL SIGNIFICANCE OF CALCIFIED PARABRONCHIAL AND PARATRACHEAL LYMPH GLANDS

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Very little direct attention has been focused on the surgical significance of calcified lymph glands producing bronchial obstruction with atelectasis, cough, hemoptysis, bronchiectasis, and bronchiolithiasis. The normal distribution of these glands in and about the tracheobronchial tree, particularly at points of bifurcation, permits pressure and bronchial obstruction by these nodes when they become diseased, with consequent enlargement or calcification. The exact mechanism of the production of calcification in lymph glands cannot always be determined. However, it is known that any dying or dead tissue surrounded by living tissue and accessible to body fluids has a tendency to take up calcium salts in the process of healing. Such changes are common following tuberculous infections, histoplasmosis, or chronic suppurative processes. Since such disorders are commonly encountered in the lungs, it is surprising that there is not an even higher incidence of calcification in the filtering tracheobronchial glands. Probably the calcification of most tracheobronchial lymph glands occurs as a result of primary tuberculous infection during childhood.

Tuberculous infection of tracheobronchial or hilar lymph glands as part of the primary complex producing atelectasis, obstructive emphysema, and subsequent bronchiectasis in the lungs of children has been not uncommonly reported in the literature.[1-4] Many instances of bronchoesophageal fistulas resulting from erosion by calcified lymph glands are recorded.[5] Also, case reports of spontaneous cures following extrusion and expectoration of the calcified contents of lymph glands are not unusual.[6] Only recently, however, has the attention of the thoracic surgeon been focused on the possibilities of the surgical management of lesions produced by obstructing inactive calcified parabronchial glands. Head and Moen,[6] in 1948, reported ten cases demonstrating some of the late nontuberculous complications of calcified hilus lymph nodes. In this series only one case of middle lobe obstruction was subjected to open thoracic operation. Buckles,[7] in 1949, suggested a more direct surgical approach to calcified hilar glands producing bronchial obstruction, and emphasized that in the past insufficient importance had been attached to the mere presence of these calcified nodes.

In many instances, the roentgenographic findings of atelectasis, along with a history of cough and hemoptysis, are very properly thought to result from

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a primary bronchiogenic malignancy until extrabronchial compression with or without erosin by calcified parabronchial lymph glands is demonstrated at operation to be the actual cause of the symptoms and findings.

Final etiological diagnoses, even following endoscopy and bronchography, are often impossible to make preoperatively because of the usual extrabronchial location of the calcified gland producing the bronchial compression. For this reason, also, it is difficult to differentiate these lesions from primary bronchiogenic carcinomas which are not uncommonly found outside of the bronchial wall and can produce bronchial compression and atelectasis without presenting within the lumen.

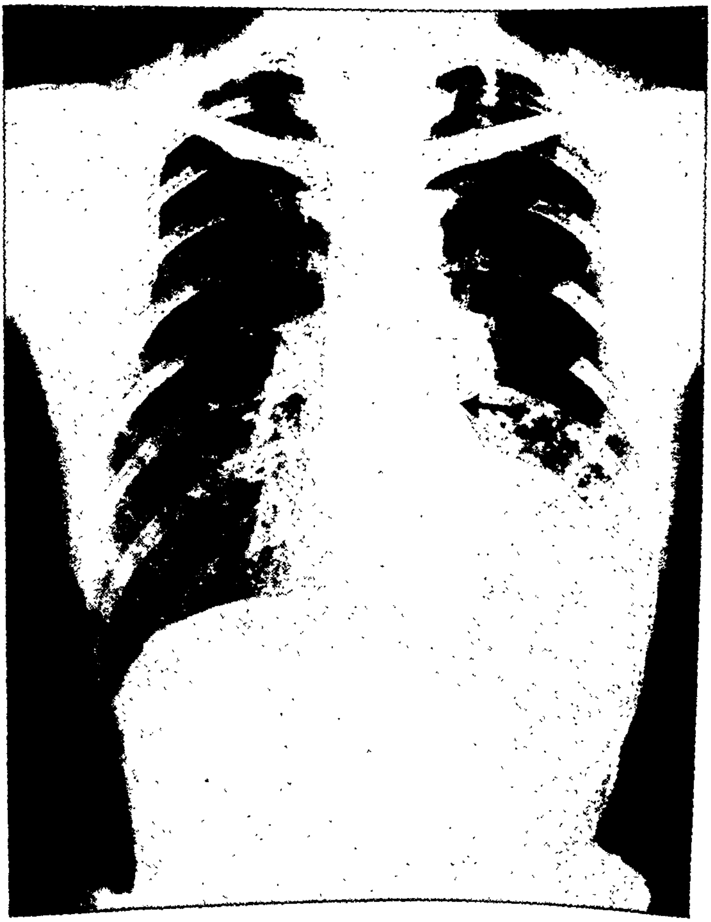

Fig. 1.—(Case 1.) Arrow points to calcification. Note atelectasis in left lower lobe.

As with many other nonmalignant lesions producing obstruction with symptoms simulating carcinoma, surgical excision of the diseased lung along with the calcified lymph glands invariably results in a cure.

To illustrate graphically the practical application of these observations, recent experiences with three patients are presented.

## CASE REPORTS

CASE 1.—M. H., a 47-year-old white woman, was admitted to Christ Hospital on Nov. 10, 1950, with a protracted four-year history of cough, hemoptysis, purulent sputum, and constantly recurring upper respiratory infections. The patient had previously been studied in another hospital where bronchographic examination had revealed bronchiectasis in the lower lobe of the left lung. At the time of admission to the local hospital the patient was running a low-grade fever and presented signs and symptoms of a pneumonic infection in the left lower lobe. Routine studies of the urine and blood revealed no abnormalities.

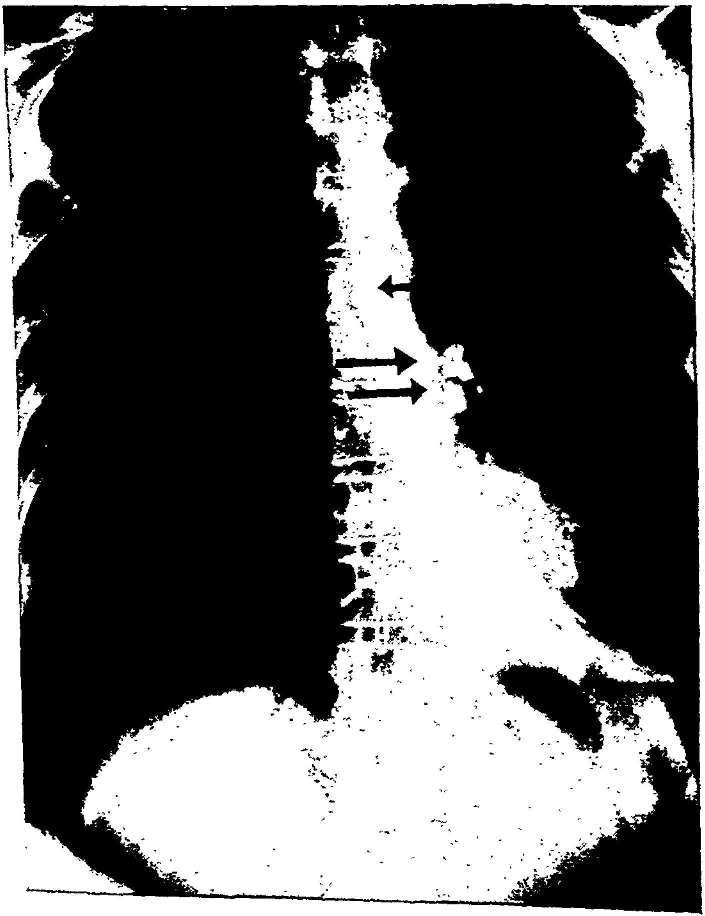

Fig. 2.—(Case 1.) Bucky film reveals three areas of calcification. Middle arrow points to calcified body within lumen of bronchus.

A posteroanterior projection roentgenogram of the chest made on admission to the hospital revealed atelectasis and pneumonia in the lower lobe of the left lung in the area in which bronchiectasis had been previously described. A small calcified lymph gland, to which little significance was attached at the time, was noted in the left paramediastinum (Fig. 1).

Bronchoscopic examination on Nov. 25, 1950, revealed what was thought to be a large, dirty-gray tumor mass arising from the wall of the left main-stem bronchus at a point one and one-half inches distal to the primary carina. The tumor was stony hard, so that a biopsy was difficult to obtain with the forceps. A crunching noise was produced when the forceps was applied. It was obvious at the time of this examination that we were dealing with a calcified lesion which was covered with epithelium and had produced almost complete obstruction of the left main-stem bronchus. Attempts to dislodge the tumor or calcified body by endoscopic manipulation were unsuccessful because of one ramification which could be seen extending into the left upper lobe orifice. Scrapings were obtained from the epithelium covering the calcified body, and on microscopic examination in the pathology laboratory, chronic inflammation and polyp formation were described. Bronchial washings were negative for acid-fast organisms and tumor cells. In the differential diagnosis, broncholith and chondroma or osteoma of the bronchus were considered.

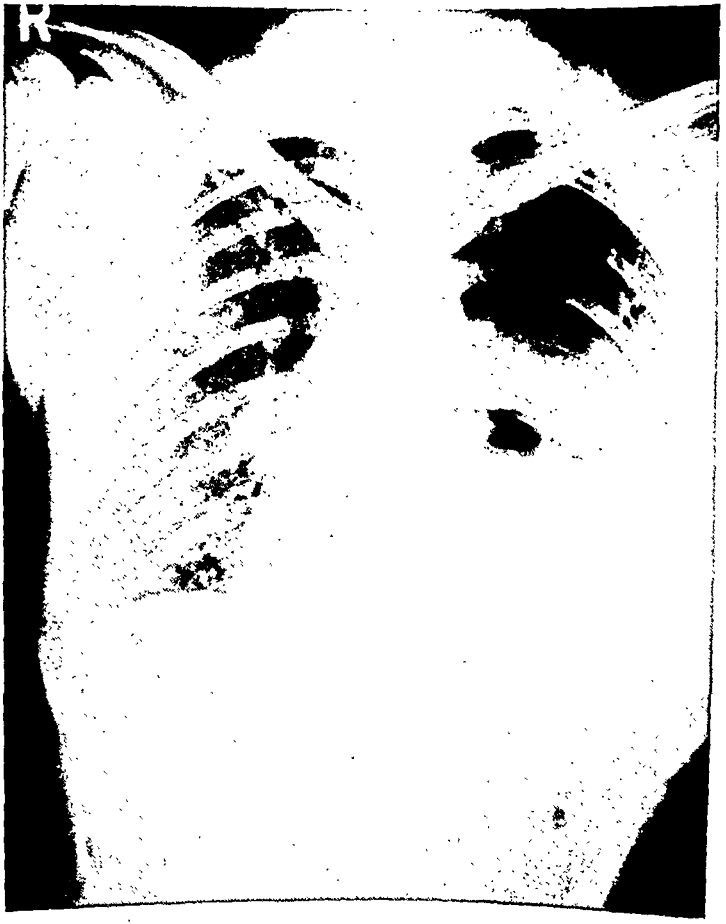

Fig. 3.—(Case 1.) Roentgenogram of chest following left pneumonectomy.

A repeat roentgenogram of the chest, posteroanterior projection, using Potter-Bucky technique, was made following the bronchoscopic examination. For the first time three calcified bodies in and about the tracheobronchial tree near the midline on the left were revealed. One calcified body was easily apparent in the lumen of the left main-stem bronchus (Fig. 2).

Because of the location of the calcified body in the left main-stem bronchus in such a position that it could not be dislodged by endoscopic manipulation, and also because of the findings of irreversible changes, namely, bronchiectasis in the lung distal to the point of obstruction, pneumonectomy was recommended.

On Dec. 1, 1950, a left pneumonectomy was performed using the classical individual vessel ligation technique. Dissection in the mediastinum was made tedious because of extensive deposits of scar tissue and the presence of several firm but noncalcified lymph glands about the main-stem bronchus. The patient did well following the operation, and was discharged approximately two and one-half weeks after admission (Fig. 3).

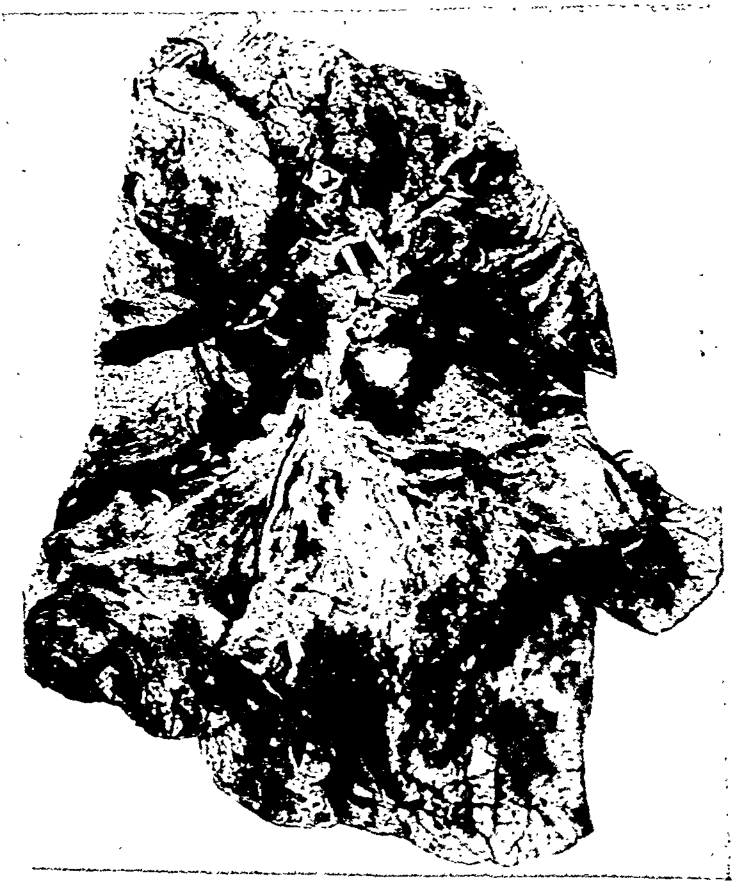

Fig. 4.—(Case 1.) Arrow points to calcification in left main-stem bronchus and anchored in orifice of left upper lobe bronchus. Note anterior calcified parabronchial lymph gland and advanced bronchiectasis in left lower lobe.

Complete dissection of the lung following surgical removal revealed no evidence of tumor. A large broncholith which had the configuration of a Staghorn calculus, such as is encountered in the pelvis of a kidney, almost completely occluded the left main-stem bronchus. One "horn," as was noted at the time of bronchoscopy, extended into the left upper lobe bronchus. A second large calcified gland was situated anterior to the left main-stem bronchus. There was advanced cylindrical and saccular bronchiectasis involving the lower lobe of the left lung (Fig. 4).

## COMMENT

The serious significance of the calcified body in the left paramediastinum was not appreciated before the bronchoscopic examination because of the relatively normal roentgenographic findings in the left upper lobe without gross evidence of atelectasis or mediastinal shift. Roentgenograms of the chest using Potter-Bucky technique made following the bronchoscopic examination, however, for the first time revealed three calcified bodies, one of which was thought to be within the lumen of the left main-stem bronchus. This woman had suffered several years with recurrent upper respiratory infections which had been diagnosed as bronchiectasis of the left lower lobe, but there had not been any real understanding of the exact cause of the disease on the left side. This experience emphasizes the importance of an endoscopic examination prior to all bronchographic studies.

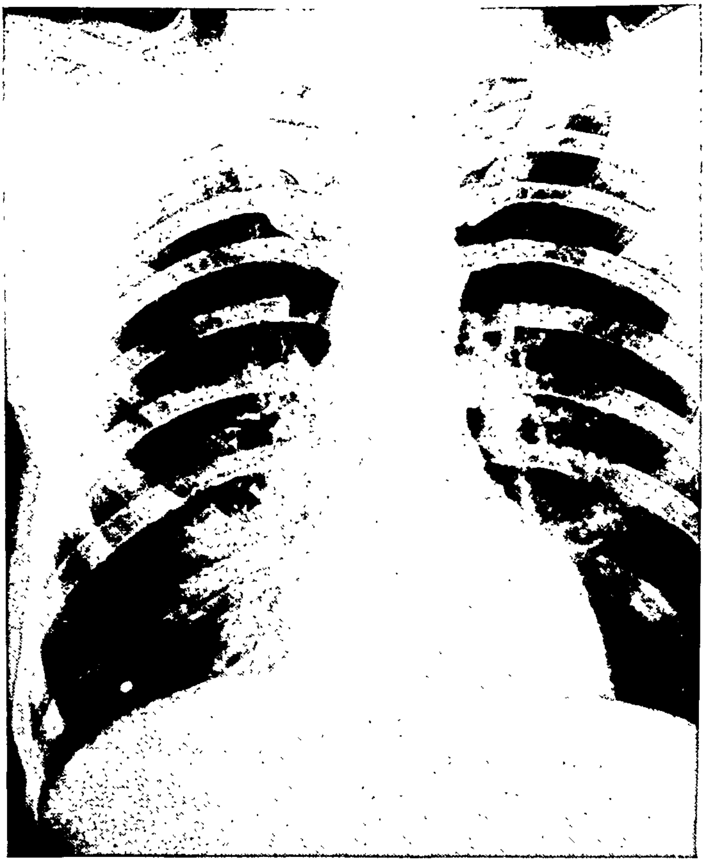

Fig. 5.—(Case 2.) Admission roentgenogram of chest. Arrows point to three calcified lymph glands. Note density right lower mid-lung field.

Case 2.—R. N., a 40-year-old white man, was admitted to Christ Hospital on Dec. 17, 1950, because of the incidental finding, by roentgenologic examination, of an infiltrate or atelectasis in the lower right mid-lung field. The patient was entirely without symptoms referable to his chest, but because of a family history of pulmonary carcinoma he was anxious to have further specialized investigation. At the time the patient was admitted to the hospital his father was dying from an inoperable carcinoma of the lung.

Roentgenograms of the chest made on admission revealed the area of infiltrate in the right lower mid-lung field which had been under observation between October and December, 1950. Particular attention was focused on what were thought to be three calcified lymph glands about the right tracheobronchial tree near the mediastnum (Fig. 5).

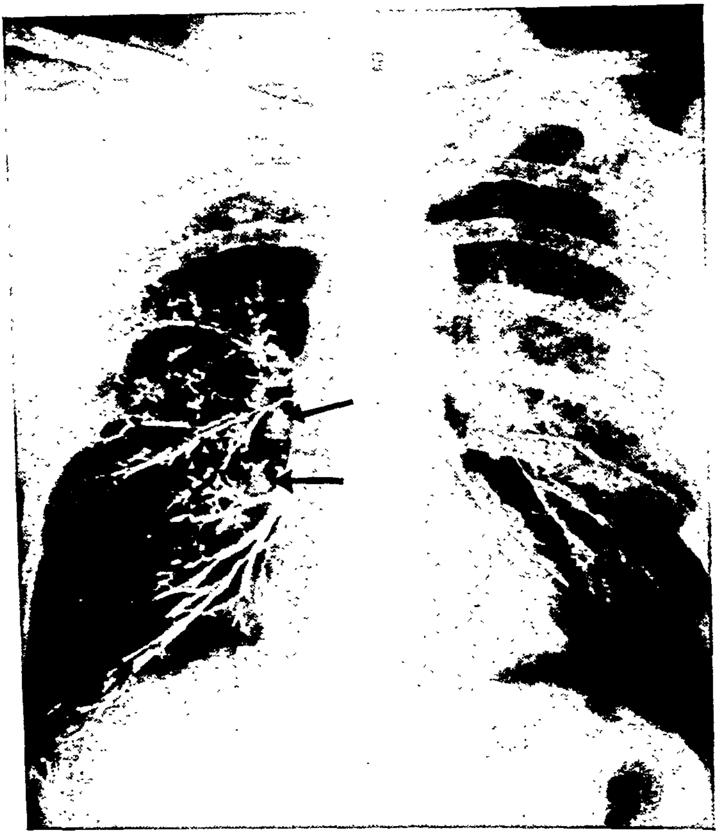

Fig. 6.—(Case 2.) Posteroanterior projection bronchogram showing relationship of calcified glands to lobar bronchi. Bronchiectasis not seen on this roentgenogram.

Bronchoscopic examination on Dec. 18, 1950, revealed entirely normal findings except for an area of pin-point erosion on the anterior wall of the right lower lobe bronchus. The orifice of the middle lobe bronchus was easily visualized and presented no evidence of obstruction or new growth. Material aspirated for cytologic study revealed atypical cells which were thought to arise from a primary malignant lung tumor. Acid-fast studies on the bronchial washings were negative for tubercle bacilli.

Bronchogram, performed following the bronchoscopic examination, revealed areas of saccular bronchiectasis involving one of the basal branch bronchi posteriorly in the right side. At the time of this examination the calcified lymph glands could be more accurately located above and below the right upper lobe bronchus and in the vicinity of the right middle lobe bronchus (Fig. 6).

A right posterior oblique projection roentgenogram again demonstrated the position of two calcified lymph glands above and below the right upper lobe bronchus, and the presence of a third calcified gland in the vicinity of the middle lobe bronchus. The middle lobe bronchus did not fill with oil despite the fact that the anterior divisions of the upper and lower lobe bronchi were well filled and adequately visualized. This excluded the possibility of improper positioning and failure of filling (Fig. 7). At the time of this examination the microscopic report on the bronchial washings was not available, and the significance of the calcified lymph glands was still not appreciated. It was our impression that we were dealing with a bronchiogenic carcinoma in the right middle lobe bronchus at a point distal to the orifice so that visualization by bronchoscopic examination was impossible. Exploratory thoracotomy was recommended.

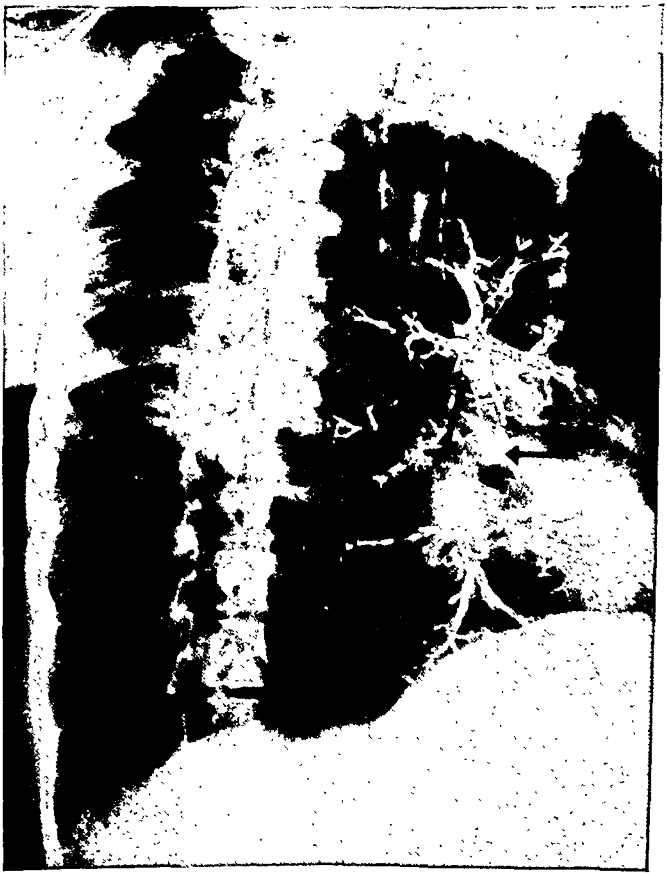

Fig. 7.—(Case 2.) Right posterior oblique projection bronchogram. Note absence of filling of right middle lobe bronchus and presence of calcified body at point of origin of right middle lobe bronchus.

Operation was performed on Jan. 16, 1951. With the patient in the face-down position, the right thorax was entered through the bed of the sixth rib which was subperiosteally resected in its entirety. The right middle lobe was completely atelectatic and replaced by firm, dense, elastic tissue. There was an irregular area of increased consistency in the posterior portion of the right lower lobe where bronchiectasis had previously been demonstrated by bronchographic study. Palpation of the central portion of the hilum of the lung revealed a large, very firm mass, approximately two to four cm. in diameter, which because of its location deep between the lobar vessels and bronchi was not accessible for direct biopsy. In view of the gross findings of atelectasis and obstruction of the middle lobe with bronchiectasis and areas of increased consolidation in the right lower lobe, as well as the findings of a mass in the vicinity of the hilum of the right lung, and previously described bronchial washings which revealed possible tumor cells, a right pneumonectomy was decided upon. Removal of the right lung was accomplished with a great deal of difficulty because of extensive scarring of the mediastinum. During the course of the dissection, two traction diverticula of the esophagus were encountered about the right main-stem bronchus. These diverticula, which were inseparably attached to the two calcified lymph glands along the main-stem bronchus, were amputated and the resultant defects in the esophagus closed with interrupted sutures of fine silk. Postoperatively, the patient did well and was discharged on his 12th postoperative day (Fig. 8).

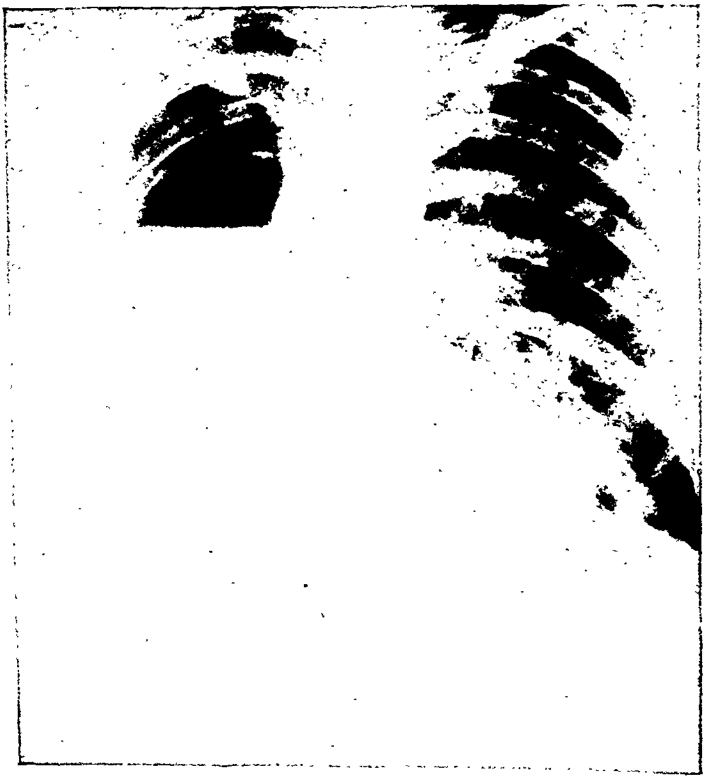

Fig. 8.—(Case 2.) Roentgenogram of chest following right pneumonectomy.

Dissection of the right lung in the pathology laboratory revealed no evidence of tumor. There was total atelectasis with far-advanced bronchiectasis and destruction of the entire middle lobe. This resulted from complete occlusion of the middle lobe bronchus by the contents of an extrabronchial lymph gland which had eroded through the wall of the bronchus and extruded into the lumen. A small pin-point erosion in the anterior wall of the basal branch bronchus with partial compression and bronchiectasis in the right lower lobe bronchus had also been produced by this same calcified extrabronchial gland. The firm mass which had been palpated at operation near the hilum of the lung corresponded in location to the extrabronchial calcified lymph gland just described. Following complete dissection of the middle lobe bronchus, one calcified body was seen to have eroded the inferior wall of this structure and then extruded into the lumen. Erosion at a second point in the opposite wall of the bronchus by pressure from the same calcified body had occurred so that it was lying in direct apposition with the pulmonary artery (Fig. 9).

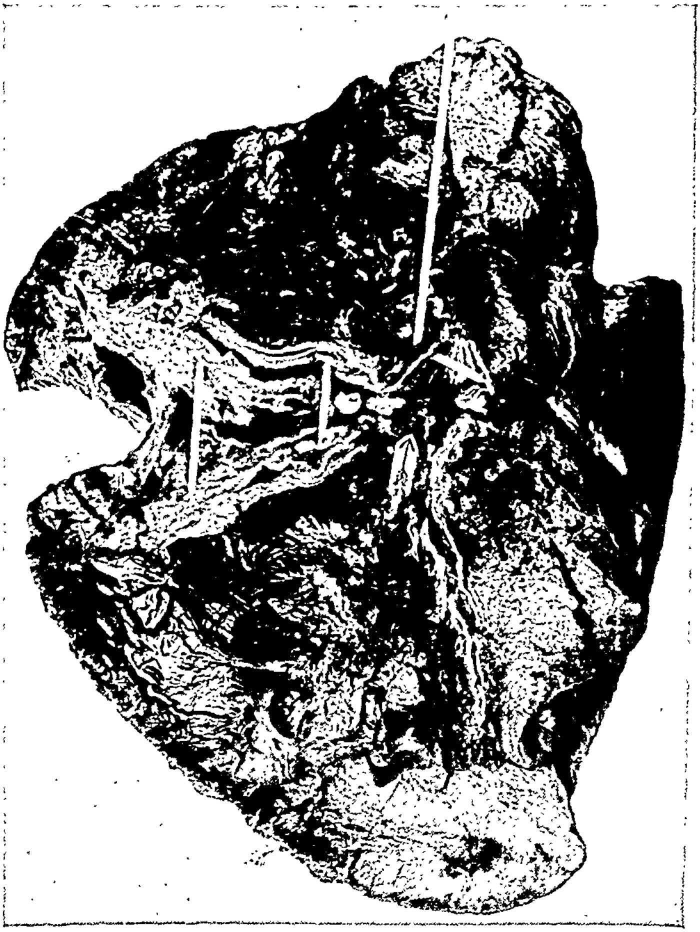

Fig. 9.—(Case 2.) Specimen of right lung. Note complete destruction of right middle lobe resulting from obstruction by calcified particles extruded into the lumen of the bronchus. Arrow points to extra bronchial calcified gland producing compression of posterior basal bronchus with bronchiectasis. Probe is in pulmonary artery.

## COMMENT

The findings in Case 2 illustrate some of the difficulties encountered in differentiating preoperatively between bronchial compression due to calcified lymph glands or carcinoma. The issue was further clouded in this particular instance because of the finding of tumor cells in the bronchial washings. Obviously, this was a false positive report. Surgical removal of the right lung obviated future difficulties which could have resulted from erosion of the pulmonary artery by the calcified body or the development of esophagobronchial fistulas if the traction diverticula of the esophagus had been permitted to go untreated.

CASE 3.—K. M., a 34-year-old white woman, was admitted to Good Samaritan Hospital on Sept. 17, 1947, with a diagnosis of tuberculoma, upper lobe, right lung. There was no history of cough, weight loss, or night sweats.

Posteroanterior projection roentgenogram of the chest, made on admission to the hospital, revealed three circumscribed, nodular, homogeneous densities, 4 cm. in diameter, in the upper lobe of the right lung (Fig. 10). At the time of this examination calcified bodies were described along the right lateral wall of the trachea, and several calcified lymph glands were noted in the vicinity of the right tracheobronchial tree just inferior to the tuberculoma. The serious significance of these findings, however, was not appreciated until the time of operation.

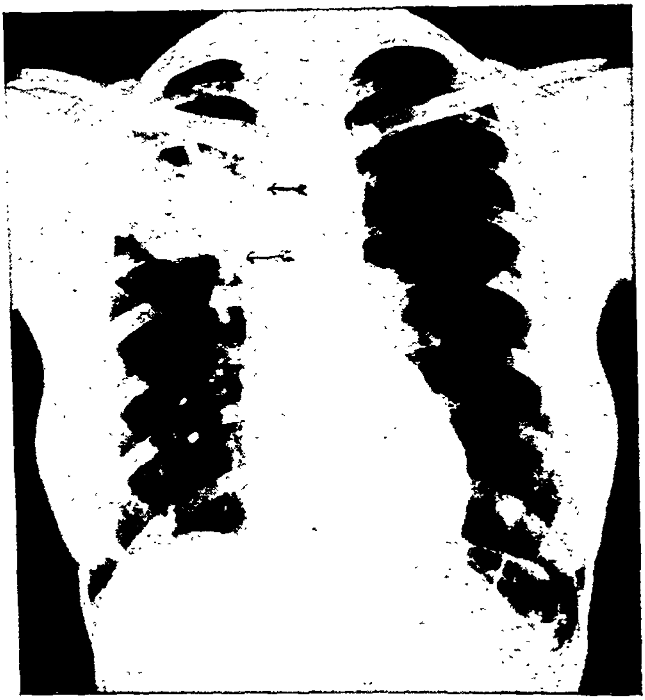

Fig. 10.—(Case 3.) Posteroanterior roentgenogram of chest showing tuberculoma of right upper lobe and calcified lymph glands along the trachea and right upper lobe bronchus.

Bronchoscopic examination on Oct. 3, 1947, revealed almost complete occlusion of the right upper lobe orifice by what appeared to be compression from an extrabronchial mass. No actual tumor or ulcer was encountered. Bronchial washings were negative for acid-fast organisms and tumor cells.

Operation was performed on Oct. 17, 1947. The upper lobe of the right lung was almost completely replaced by firm elastic tissue, the central portion of which obviously contained fluid or pus. A right upper lobectomy which had previously been scheduled was found impossible because of the complete encirclement of the right upper lobe bronchus by firm calcified lymph glands which were deeply embedded in the wall of this structure. After a prolonged unsuccessful effort at dissection of these glands, pneumonectomy was decided upon. Even this operation was found to be almost impossible to complete because of extensive calcification of the membranous wall of the main-stem bronchus and trachea with numerous calcified lymph glands inseparably embedded in the walls of these structures. One large accumulation of calcium presented in the mediastinum and was densely adherent to the pericardium. Dissection of the pulmonary vessels was made unusually difficult because of scarring in and about the mediastinum. After extensive dissection, an "S" shaped line of amputation across the right main-stem bronchus was finally developed and extended into the carina. This line of amputation passed between several calcified lymph glands, leaving just sufficient bronchial tissue so that good closure of the stump could be obtained with interrupted sutures of fine silk (Fig. 11). The calcified mediastinal plaque and a portion of the pericardium were removed with the right lung. Postoperatively, the patient did well and was discharged two weeks after operation.

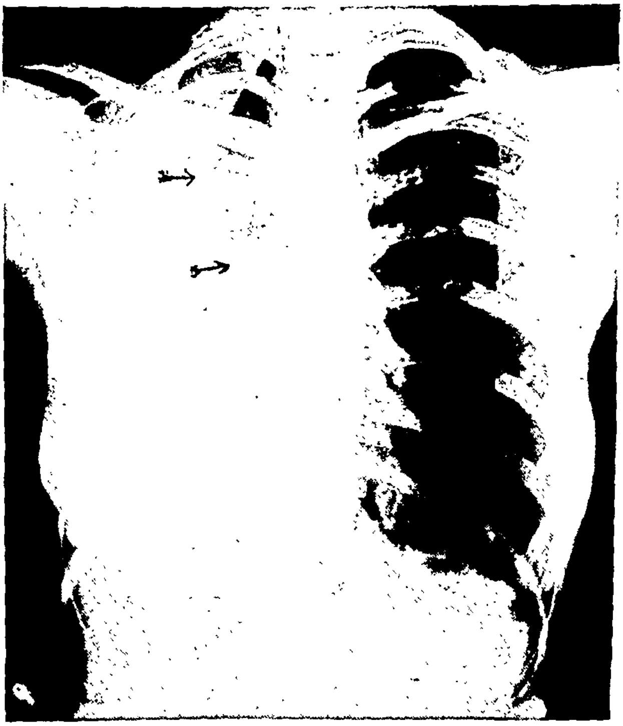

Fig. 11.—(Case 3.) Posteroanterior roentgenogram of chest following right pneumonectomy. Note calcified lymph glands along bronchial amputation stump and trachea.

Section of the lung showed the upper lobe to be occupied by several large intercommunicating thick-wall abscesses containing thick green viscid pus. Acid-fast organisms were demonstrated both on direct smear and culture of the pus contained in the abscesses.

## COMMENT

The serious importance of the calcified lymph glands seen on the roentgenograms of the chest in Case 3 was not realized prior to operation. The mere anatomic presence of these glands made necessary a radical complete pneumonectomy instead of a simple right upper lobectomy in the management of a benign tuberculoma.

## DISCUSSION

The serious significance of the presence of calcified lymph glands in and about the tracheobronchial tree probably has not received sufficient emphasis in the past. In patients with isolated areas of atelectasis due to bronchial compression by calcified lymph glands, the clinical history, physical findings, roentgenographic changes, and even endoscopic and bronchographic studies can be identical with the findings encountered in patients with atelectasis which is due to a peripherally located or extrabronchial pulmonary carcinoma.

The third case report illustrates the major surgical consideration an operator may be forced into by the incidental finding at the time of operation of deeply embedded calcified parabronchial lymph glands. The anatomic position of these calcified bodies about lobar bronchi may obviate adequate dissection and stump closure at this level and force the operator to amputate much higher with sacrifice of normal lung tissue.

Valuable information about the presence of unsuspected calcified lymph glands and their relationship to the tracheobronchial tree can be obtained preoperatively by more liberal use of heavy exposure roentgenograms and laminagrams of the chest in cases where the exact cause of the atelectasis or abnormal infiltrate is not immediately apparent.

## CONCLUSIONS

1. The serious significance of calcified lymph glands is not always appreciated until open thoracic operations show these structures to be the real cause of lung pathology, the exact etiology of which was originally obscure.
2. The finding of calcified lymph glands by roentgenogram in and about the tracheobronchial tree is usually associated with dense scarring of the mediastinal and interlobar structures, and difficult surgical dissection at the time of operation can be anticipated.
3. The mere presence of nonobstructing calcified lymph glands about the lobar bronchi may necessitate the performance of more radical procedures than were preoperatively anticipated to remove lesions for which more conservative surgery is ordinarily recommended.
4. Extrabronchial compression, with or without erosion by calcified lymph glands, can produce all of the symptoms and roentgenologic findings of primary pulmonary carcinoma.
5. As with other nonmalignant lesions producing bronchial obstruction, complete excision of the destroyed portions of lung with the calcified lymph gland will invariably result in a cure.
6. More careful preoperative roentgenologic studies of the chest using Potter-Bucky techniques and laminagrams are invaluable in demonstrating the presence of calcified lymph glands and their relationship to portions of the tracheobronchial tree which might be obstructed by compression.

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## Original Communications

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# OPEN PNEUMONOLYSIS—ITS VALUE AND LIMITATIONS

## A Review of Thirty-six Operated Cases

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OPEN intrapleural pneumonolysis is an old procedure. As early as 1908 Friedrich performed the first operation of this kind. The result, however, was not very encouraging. When division of adhesions was attempted, cavity rupture occurred, and the patient died shortly afterward. In Sweden, open pneumonolysis was first performed by Key, in cooperation with Jacobaeus, in 1915. To obtain sufficient working space, two ribs were resected and the lung was then freed extrapleurally, a procedure which was later recommended by many surgeons. Open pneumonolysis, however, never obtained general favor. Poor results, chiefly attributable to complicating specific and nonspecific empyemas, accentuated the lack of enthusiasm. Moreover, the field of indications became more sharply circumscribed by the introduction of Jacobaeus' closed intrapleural pneumonolysis operation, and by the development of thoracoplasty technique. According to Kremer, only 100 open, as compared to 5,000 closed pneumonolyses, were performed before 1938.

In America, meanwhile, there were advocates of the open method. It even happened that open pneumonolysis completely supplanted the closed procedure. Certainly Alexander, in Ann Arbor, who is a pioneer in this field, stated in 1925 that "thoracoplasty is preferable to pneumothorax which must be preceded by so risky an operation as open intrapleural pneumonolysis." But further experience led him, in 1937, to express some degree of alteration in his views. He said then, "developments in the technique of open intrapleural pneumonolysis have made the operation a valuable one for a small group of patients." In the same year, Alexander reported a series of his own, comprising twenty-nine cases, and reviewed the experiences of certain other American surgeons.

When extrapleural pneumothorax was thereafter introduced as a therapeutic measure, communication with the pleural cavity sometimes arose when pneumonolysis was carried out. The collapse thus obtained is designated "pneumothorax mixte." This gave to many workers an increased comprehension of how the pleura in tuberculosis reacts when it is extensively opened. As regards

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collapse, no fundamental difference is implied if, on inducing extrapleural pneumothorax, free communication with the pleural cavity is obtained, or if the pleura is first opened and the lung thereafter freed extrapleurally, which is what commonly happens. Holst stated also in 1946: "If one wishes to enter the extrapleural space directly as in extrapleural pneumothorax or transpleurally through a free pleural cavity is a question of choice of technical method in each individual case."

The early 1940's saw a reawakening in Sweden of the dormant interest in open pneumonolysis. The operation was taken up by one of the writers (Crafoord) in cooperation with various other phthisiologists (Dahlstedt, Gullbring, Westergren) and by Hultén in cooperation with Hedvall. In 1947 Crafoord reported fifty-five, and Hultén seventy-three operated cases. Hedvall spoke in 1946 of "wide-open" pneumonolysis as a new surgical method in tuberculosis therapy. Hulten stated at the same time: "Aftercare is of fundamental importance, and there apply the innovations which we have reported." Modern postoperative treatment of the pleural cavity with penicillin has certainly reduced the risk of secondary infection formerly so dreaded in these patients. Such enthusiasm for, and wide experience of open pneumonolysis as that of Hedvall and Hultén are probably not to be found elsewhere is this country; but Swedish phthisiologists and surgeons now recognize the procedure, and employ it sometimes in cases which might otherwise have been considered for closed division of adhesions or thoracoplasty.

## PERSONAL INVESTIGATIONS

The following is a report of our joint series of thirty-six patients treated with open intrapleural pneumonolysis. One of us (Crafoord) has, in cooperation with other phthisiologists, performed the operation on a further forty patients up to 1949. *To open pneumonolysis have been referred cases where (a) closed division of adhesions might possibly have been considered but found inadvisable, (b) artificial pneumothorax would otherwise have been terminated in favor of thoracoplasty* or, in a few instances, extrapleural pneumothorax.

The distribution of the clinical material over calendar years is:

|  | year | 1943 | 1944 | 1945 | 1946 | 1947 | 1948 | 1949 |
|---|---|---|---|---|---|---|---|---|
| Number of cases |  | 1 | 1 | 14 | 8 | 4 | 4 | 4 |

It is seen that the majority of the operations were performed in 1945 and 1946, the years following Hedvall's and Hultén's report on their preliminary results. It may, even at this point, be mentioned, and should be in the nature of things, that indications change as experience of the method increases. For this reason our series has fewer patients after 1947. As final results after voluntary cessation of pneumothorax therapy can be presented only for those patients whose operations were performed up to and including 1946, *the material has been divided into two main groups. Group A comprises the period 1943-1946 and consists of twenty-four patients. Group B includes 1947-1949, and has only twelve patients.*

The most important clinical details of each patients are reported in Table I.

*Group A* (1943-1946).—

The twenty-four patients in this group are most conveniently divided into (1) those with *cord-type adhesions*, where open pneumonolysis was carried out instead of the closed procedure, and (2) patients with *direct fusion of the* visceral and parietal *pleurae*, where open pneumonolysis was substituted for thoracoplasty (or possibly extrapleural pneumothorax). The former subgroup contains four and the latter twenty patients. Open pneumonolysis in both these adhesion types is discussed below.

*Cord-type Adhesions*.—In the group of four cases where open pneumonolysis was substituted for closed, short, thick cords attached to the chest wall held pulmonary cavities distended. Cavity diameters did not exceed 3 cm. All these patients were sputum positive before operation. Rapid postoperative cavity closure was obtained in all four cases, and therapeutic pneumothorax could be successfully maintained. Serous effusion occurred in only one case, and was reabsorbed spontaneously. When air refills were discontinued, clinical cavity closure was present in all cases. In three patients the treated lung showed only insignificant failure to re-expand; in the fourth there was conspicuous ipsilateral inexpansibility with fixation of the diaphragm. As this is not infrequently a sequel to pneumothorax therapy where open pneumonolysis has not been carried out, the *final results* must be regarded as *satisfactory*.

*Direct Fusion of the Pleurae*.—*Preoperative period:* In twelve of the twenty cases where open pneumonolysis was substituted for thoracoplasty (or possibly extrapleural pneumothorax) the untreated lung was healthy. Of the remaining eight patients in this group, two had simultaneous contralateral pneumothorax; a further two had reduced contralateral capacity following concluded pneumothorax and thoracoplasty, respectively. The untreated lung presented concomitant lesions, relatively insignificant in extent in three patients, and in one, cavernous tuberculosis.

In all twenty patients, there was cavernous tuberculosis of the pneumonolysis-treated lung. The respective diameters of cavities were approximately 1 cm. in five patients, 2 cm. in four, 3 cm. in six, and 4 cm. in five cases. With only one exception the entire series was sputum positive on direct smears prior to pneumothorax induction. Seventeen patients were still sputum positive at the time of open pneumonolysis.

More or less extensive closed division of adhesions had already been carried out in seven cases; thirteen were thus referred to open pneumonolysis directly or after thoracoscopy. Pleural effusion was present preoperatively in seven patients; in six of these the exudate was serous, although *M. tuberculosis* could be cultured. One patient had tuberculous empyema.

*Operation:* Direct symphysis was chiefly localized to the pleurae in the region of the cupola and the mediastinal surface, and varied in extent; but in most cases the area freed was at least as large as the palm of the hand. Sometimes the adhesion was loose but as a rule stripping was undertaken extrapleurally in thickened, sometimes particularly dense tissue. The process of stripping was often associated with considerable difficulty.

TABLE I

| CASE NO. | SEX | AGE | PREOPERATIVE PERIOD | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | RIGHT LUNG | LEFT LUNG | SPUTUM | PNEUMOTHORAX INDUCED | THORACOSCOPY; CLOSED PNEUMONOLYSIS | EFFUSION |
| 1 | Female | 28 | Ap.–$I_2$. Cav. 2 cm. $I_2$. | Nothing abnormal | Pos. | 12- 2-43 | Partial cl. pnml. | — |
| 2 | Female | 38 | Cav. 1 cm. $I_2$. | Pnth. 33-40. | Pos. | 7- 5-43 | — | June, '44; Empyema, Oct. 44 |
| 3 | Male | 24 | Upper lobe. Cav. 3 cm. $I_1$. | Nothing abnormal | Pos. | 30-11-44 | — | Serous Jan., '45 |
| 4 | Male | 37 | Upper lobe. Cav. 4 cm. $I_2$. | Nothing abnormal | Pos. | 7-10-44 | Thscpy. 0 cl. pnml. | — |
| 5 | Male | 47 | Upper lobe. Cav. Ap.–$I_2$, max. 4 cm. | Insignificant $I_{2-4}$. | Pos. | 10- 1-45 | Thscpy. 0 cl. pnml. | — |
| 6 | Male | 32 | Nothing abnormal | Ap.–$I_1$. Cav. 2 cm. Ap. | Pos. | 13- 2-45 | Thscpy. 0 cl. pnml. | — |
| 7 | Male | 31 | Nothing abnormal | Ap.–$I_2$. Cav. 2 cm. $I_1$. | Pos. | 25-10-44 | Thscpy. 0 cl. pnml. | — |
| 8 | Female | 25 | Ap.–$I_1$. Cav. 3 cm. Ap. | Nothing abnormal | Neg. | 29- 4-45 | Thscpy. 0 cl. pnml. | Serous June, '45 |
| 9 | Female | 25 | Upper lobe. Cav.$I_{1-2}$, max. 3 cm. | Nothing abnormal | Pos. | 10- 3-44 | Partial cl. pnml. | — |
| 10 | Female | 43 | Ap.–$I_2$. Cav. 3 cm. $I_1$. | Nothing abnormal | Pos. | 16-10-44 | Partial cl. pnml. | — |
| 11 | Male | 25 | Ap.–$I_2$. Cav. 2 cm. Ap. | Apical thoracoplasty '42 | Pos. | 16- 6-45 | Partial cl. pnml. | — |
| 12 | Male | 20 | Pnth. since April '43. | Upper lobe. Cav. Ap.–$I_1$, max. 4 cm. | Pos. | 11- 6-45 | Partial cl. pnml. | — |
| 13 | Male | 52 | Nothing abnormal | Ap.–$I_3$. Cav. 2 cm. $I_1$. | Pos. | 11- 8-45 | Partial cl. pnml. | — |
| 14 | Female | 34 | Ap.–$I_1$. Cav. 2 cm. $I_1$. | Nothing abnormal | Pos. | 3- 4-45 | Partial cl. pnml. | Serous July, '45 |
| 15 | Female | 33 | Nothing abnormal | Ap.–$I_2$. Cav. 1 cm. $I_1$. | Pos. | 1- 9-45 | Thscpy. 0 cl. pnml. | Serous Oct., '45 |
| 16 | Female | 33 | $I_{1-2}$. Cav. 1 cm. $I_1$. | Nothing abnormal | Pos. | 2- 7-45 | — | Serous July, '45 |
| 17 | Male | 22 | Nothing abnormal | Upper lobe. Cav. 2 cm. $I_1$. | Pos. | 5- 9-45 | Thscpy. 0 cl. pnml. | — |
| 18 | Female | 28 | Upper lobe. Cav. 3 cm. $I_1$. | Small cav. Ap. lower lobe | Pos. | 25-10-45 | Partial cl. pnml. | — |

| OPERATION | | POSTOPERATIVE PERIOD | | | FINAL RESULTS | | |
|---|---|---|---|---|---|---|---|
| DATE | EXTENT | IMMEDIATE COMPLICATIONS | EFFUSION, EMPYEMA | CAVITY CLOSURE | PNEUMOTHORAX TERMINATED | CAVITY HEALING | PULMONARY INEXPANSIBILITY |
| 5/ 7/43 | Index-finger-thick cord, some mm. long. | — | — | Yes | 1945 | Yes | Insignificant |
| 4/11/44 | Direct pleural fusions 4 cm. | — | Empyema, see PREOPERATIVE PERIOD | Yes | 1947 | Yes | Extreme |
| 3/ 2/45 | Extensive direct fusion. | — | Empyema Sept., '45. Oleothorax | Yes | See EFFUSION, EMPYEMA | Yes | Moderate |
| 0/ 3/45 | Direct medial pleural fusion: Ap.-Hilus. | — | Serous effusion | Yes | 1948 | Yes | Extreme |
| 7/ 4/45 | Extensive coarse direct pleural fusion. | — | Serous effusion | No | Bilateral cavities. Died, 47 | — | — |
| 17/ 4/45 | Direct pleural fusions mediastinally. | — | Serous effusion. Empyema, '46. | Yes | 1949 | Yes | Extreme |
| 25/ 4/45 | Finger-thick short cord. | — | — | Yes | 1949 | Yes | Insignificant |
| 10/ 7/45 | Direct fusion, the size of a child's palm. | — | Effusion, see PREOPERATIVE PERIOD. No empyema. | Yes | 1950 | Yes | Extreme |
| 1/ 8/45 | Direct fusion, the size of a child's palm. | — | Serous effusion. Empyema, '46. | Yes | 1948 | Yes | Moderate |
| 13/ 9/45 | Finger-thick cord, some mm. long. | — | — | Yes | 1949 | Yes | Moderate |
| 13/10/45 | Direct pleural fusions mediast. Ap.-Hilus (loose). | — | Serous effusion, insignificant. | Yes | 1948 | Yes | Moderate |
| 23/10/45 | Direct pleural fusions 4 cm. | Cav. perf. 18-11-45. Mixed-infect.. Empyema. Died 10-12-45 | — | — | — | — | — |
| 3/11/45 | Direct pleural fusions 5 cm. | — | Serous effusion, insignificant. | Yes | 1949 | Yes | Insignificant |
| 10/11/45 | Direct thick fusion, the size of a child's palm | — | Effusion, see PREOPERATIVE PERIOD. Empyema, '46. | Yes | 1949 | Yes | Moderate |
| 6/12/45 | Extensive direct pleural fusions, the largest the size of the palm. | — | Effusion, see PREOPERATIVE PERIOD. No empyema. | Yes | 1949 | Yes | Extreme |
| 17/12/45 | Extensive direct pleural fusions. | — | — | Yes | 1950 | Yes | Extreme |
| 16/ 2/46 | Short cords. | — | Serous effusion, insignificant. | Yes | 1950 | Yes | Insignificant |
| 23/ 2/46 | Extensive firm direct fusions. | Mil. tub. Died 25-3-46 | — | — | — | — | — |

TABLE I

| CASE NO. | SEX | AGE | PREOPERATIVE PERIOD | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | RIGHT LUNG | LEFT LUNG | SPUTUM | PNEUMOTHORAX INDUCED | THORACOSCOPY: CLOSED PNEUMONOLYSIS | EFFUSION |
| 19 | Male | 25 | Nothing abnormal | Ap.–$I_1$. Cav. 1 cm. $I_1$. | Pos. | 17- 1-46 | — | — |
| 20 | Male | 21 | $I_{1-3}$. Cav. 1 cm. $I_1$. | Pnth. since Sept. '42. | Pos. | 5- 9-44 (no adhesions) | Thscpy. 6-3-46. Dir. pl. fus. upper lobe. Sput. pos. | — |
| 21 | Male | 54 | Ap.–$I_3$. Cav. 3 cm. $I_1$. | Ap.–$I_2$. No cav. | Pos. | 15- 6-46 | 0 thscpy. Cl. pnml. | — |
| 22 | Female | 45 | Ap.–$I_2$. No cav. | Ap.–$I_3$. Cav. 4 cm. $I_1$. | Pos. | 29- 1-46 | Thscpy. 0 cl. pnml. | — |
| 23 | Male | 31 | Upper lobe. Cav. 3 cm. Ap. | Nothing abnormal | Pos. | 15- 8-46 | Partial cl. pnml. | Serous Sept., '46 |
| 24 | Male | 46 | Ap.–$I_4$. Cav. Ap.–$I_2$, max. 3 cm. | Nothing abnormal | Pos. | 15- 8-46 | Thscpy. 0 cl. pnml. | — |
| 25 | Female | 33 | Upper lobe. Cav. 3 cm. Ap. | Nothing abnormal | Pos. | 31- 7-46 | Partial cl. pnml. | — |
| 26 | Female | 38 | Upper lobe. Cav. 3 cm. $I_1$. | Nothing abnormal | Pos. | 11- 3-47 | Partial cl. pnml. | Serous Apr., '47 |
| 27 | Female | 40 | Upper lobe. Cav. 3 cm. $I_1$. | Nothing abnormal | Pos. | 8- 5-47 | Partial cl. pnml. | Serous May, '47 |
| 28 | Female | 27 | Ap.–$I_2$. No cav. | Upper lobe. Cav. 3 cm. $I_1$. | Pos. | 3- 3-47 | Partial cl. pnml. | — |
| 29 | Male | 32 | Upper lobe. Cav. 4 cm. $I_1$. | Nothing abnormal | Pos. | 11-10-47 | Thscpy. 0 cl. pnml. | — |
| 30 | Male | 46 | Nothing abnormal | Upper lobe. Cav. 2 cm. $I_1$. | Pos. | 27-10-47 | Partial cl. pnml. | — |
| 31 | Male | 24 | Nothing abnormal | Ap.–$I_5$. Cav. $I_{1-2}$, max. 4 cm. | Pos. | 10- 6-48 | Thscpy. 0 cl. pnml. | Serous July, '48 |
| 32 | Male | 37 | Upper lobe. Cav. 4 cm. $I_1$. | Insignificant $I_{3-4}$. | Pos. | 18- 8-48 | Thscpy. 0 cl. pnml. | — |
| 33 | Female | 31 | $I_{1-2}$. No cav. | $I_{2-5}$. Cav. $I_{2-4}$, max. 3 cm. | Pos. | 18- 8-48 | Thscpy. 0 cl. pnml. | — |
| 34 | Male | 38 | Nothing abnormal | Ap.–$I_2$. Cav. 3 cm. $I_2$. | Pos. | 11- 3-49 | Partial cl. pnml. | — |
| 35 | Male | 32 | Nothing abnormal | Ap.–$I_4$. Cav. 4 cm. Ap. | Pos. | 1- 6-49 | Partial cl. pnml. | — |
| 36 | Male | 36 | Nothing abnormal | Upper lobe. Cav. 2 cm. $I_1$. | Pos. | 5- 7-49 | Partial cl. pnml. | — |

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| OPERATION | | POSTOPERATIVE PERIOD | | | FINAL RESULTS | | |
|---|---|---|---|---|---|---|---|
| DATE | EXTENT | IMMEDIATE COMPLICATIONS | EFFUSION, EMPYEMA | CAVITY CLOSURE | PNEUMOTHORAX TERMINATED | CAVITY HEALING | PULMONARY INEXPANSIBILITY |
| 3/ 3/46 | Direct pleural fusions Ap.; The adherent lower lobe was not freed | — | Serous effusion, insignificant. | Yes | 1949 | Yes | Insignificant |
| 3/ 4/46 | Extensive. Loose direct pleural fusions. | May, '46, progress left lung | Serous effusion, April, '46. | Yes | See IMMEDIATE COMPLICATIONS. Died 26-10-46 | — | — |
| 3/ 7/46 | Palm-size, firm direct pleural fusion. | — | Serous effusion. Empyema, 47. | Yes | 1950 | Yes | Moderate |
| 23/ 7/46 | Extensive coarse direct pleural fusions. | — | Serous effusion. | Yes | 1949 | Yes | Extreme |
| 25/10/46 | Palm-size, coarse direct pleural fusion. | — | Effusion, see PREOPERATIVE PERIOD. No Empyema. | Yes | 1950 | Yes | Extreme |
| 12/11 '46 | Extensive coarse direct pleural fusions. | Hemothorax ÷ mediast. emphysema. Died 14-11-46 | — | — | — | — | — |
| 15/ 3/47 | Finger-thick cord, some mm. long. | — | — | Yes | — | — | — |
| 18/ 6/47 | Palm-size, direct pleural fusion. | — | Effusion, see PREOPERATIVE PERIOD. Empyema, '47. | Yes | — | — | — |
| 18/ 6/47 | Little-finger thick, short cord. | — | Effusion, see PREOPERATIVE PERIOD. Empyema, '47. | Bronch-pl. fistula, '50 | — | — | — |
| 5/ 7/47 | Direct pleural fusion 4 cm. | — | Serous effusion, insignificant. | No. Progr. right l. cav., '48 | — | — | — |
| 6/ 2/48 | Extensive coarse direct pleural fusions. | — | Serous effusion. Empyema, '48. | Yes | — | — | — |
| 17/ 4/48 | Extensive coarse direct pleural fusions. | Evac. of clots ÷ decortication 27-4-48 | Serous effusion. | Yes | — | — | — |
| 7/ 8/48 | Direct pleural fusion 4 cm. | — | Effusion, see PREOPERATIVE PERIOD. Empyema. Jan. '49. | Yes | — | — | — |
| 4/12/48 | Direct pleural fusion 5 cm. | — | Serous effusion | Yes | — | — | — |
| 30/ 4/49 | Extensive direct pleural fusions. | — | Serous effusion | Yes | — | — | — |
| 14/ 5/49 | Palm-size, firm direct pleural fusion. | — | Effusion, insignificant | Yes | — | — | — |
| 25/ 7/49 | Short cords. | — | Effusion, insignificant | Yes | — | — | — |
| 1/10/49 | Finger-thick cord, some mm. long. | — | Effusion, insignificant | Yes | — | — | — |

*Postoperative period:* Three patients died shortly after surgery. In one of these, death occurred after two days as a result of hemothorax and mediastinal emphysema; the second patient became suddenly very ill three weeks after operation, and died after a further three weeks from mixed-infection empyema, which was a sequel to extensive cavity perforation; there was marked postoperative deterioriation in the condition of the third patient, who died one month later from miliary tuberculosis.

Pleural effusion was present at the time of operation in seven of the remaining seventeen patients in this group. There was no reaccumulation of fluid in one case, but in the others effusion continued. Tuberculous empyema developed in 3 instances, exclusive of the case where purulent effusion was present preoperatively. In seven of the other ten patients there were large postoperative accumulations of fluid, which developed into empyema in three cases. The remaining three patients had only insignificant effusions which reabsorbed spontaneously. Persistent effusion was treated with repeated aspiration, and in two cases empyematous fluid was partly replaced by oil. Effusion finally ceased in some cases; in others, the effusion formed a covering layer at the conclusion of pneumothorax therapy. In none of these cases was there secondary pyogenic infection of the pleural cavity.

Cavity closure was obtained in sixteen patients. Bronchial tuberculosis developed in the later course of the disease in one case; both lungs were affected preoperatively in this patient, and bilateral cavernous phthisis was present at death. In another case, despite ipsilateral cavity closure, death occurred six months after surgery from hemoptysis which originated in the contralateral lesions. Thus five patients in all died during the pneumothorax period.

*Final Results.*—At the conclusion of pneumothorax therapy, this group consisted of fifteen patients. As far as can be judged, clinical healing of the tuberculous pulmonary process took place in all cases. Similarly satisfactory results cannot, however, be recorded from the functional aspect. The effusions caused pleural thickening with diaphragm fixation and pulmonary inexpansibility. In three patients, the treated lung must be regarded as functionally useless and incapable of re-expansion; preoperative empyema was present in one of these cases. The lung is still capable of aeration in another five cases, but pleural thickening is so extensive and reduction of diaphragmatic mobility so marked that the remaining function can scarcely be allotted practical importance. Yet another five patients have more moderate pulmonary inexpansibility, but such restriction of diaphragmatic movement that ventilation capacity is certainly gravely impaired. Finally, the expansile powers of the lung are only slightly reduced and diaphragmatic mobility insignificantly affected in two cases.

*Conclusions from Group A.—The account of the above series of open pneumonolysis in pneumothorax with direct fusion of the pleurae shows a not insignificant primary mortality; pleural complications, lengthy and difficult to treat, cause considerable reduction of functioning parenchyma even when satisfactory healing of lesions is obtained.* When these facts became apparent, we attempted a re-evaluation of the advisability of open pneumonolysis. We have also judged afresh in retrospect the situation in the twenty reported cases at the time of operation. In the light of experience gained, we have reconsidered whether open pneumonolysis should have been substituted for thoracoplasty. It may be pertinent to state first that two patients who declined the thoracoplasty originally advised, underwent open pneumonolysis instead; both these patients, who had pleural effusion at the time of operation, belong, of course, a priori, to the thoracoplasty group. For eight patients who had lesions of a type and extent which promised adequate collapse with a minor or moderately large thoracoplasty, this should have been the operation of choice. In six cases where subtotal or total thoracoplasty was necessary for satisfactory collapse, the choice of surgery was more difficult. The several stages of thoracoplasty constituted a disadvantage in this group. although even this did not perhaps fully outweigh the greater risk of open pneumonolysis. Other arguments for and against both procedures could certainly be presented here and the final choice of method may involve a careful analysis of all the relevant factors. Open pneumonolysis was, even in retrospect. the most suitable intervention for two patients. In one of these, local direct pleural symphysis had occurred under effective pneumothorax therapy, but was estimated to be fairly loose. In the other, the intention was to free only the diseased upper lobe, not the unaffected lower lobe which was extensively adherent; the intervention here was thus designed to produce collapse similar to that in extrapleural pneumothorax, with good diaphragmatic mobility. Finally, the general condition of two patients was so poor that the decision as to active collapse therapy should have been postponed; thoracoplasty might conceivably have been contemplated later but open pneumonolysis, which at first appeared justifiable was not, in the light of present experience, to be recommended.

It is apparent from the above discussion that, when the question of the most suitable collapse measures for the twenty reported pneumothorax patients with direct pleural fusion is reviewed against the background of our present experience, open pneumonolysis could have been advised for at most eight patients.

*Group B* (1947-1949).—

As experience of open pneumonolysis was gained, indications were applied more cautiously. Even if knowledge of the operation was as yet incomplete, the extent of its practice was decreased after 1947. Thus, no more than twelve patients were submitted to the procedure from 1947-1949 inclusive (see Table I). The period of pneumothorax is nearing conclusion in the autumn of 1950 only in the earliest operated of these patients. Final results cannot, therefore, be presented, but the more immediate sequellae to surgery may be surveyed.

Distribution of this group analogous to that of group A shows that four patients underwent open instead of closed pneumonolysis. Open pneumonolysis was substituted for thoracoplasty in eight cases.

*Cord-type Adhesions.*—All four patients upon whom open instead of closed pneumonolysis was performed were sputum positive at operation and had patent cavities. The diameters of cavities did not exceed 4 cm. The postoperative course was uneventful in three patients. In the fourth case, a short adhesion, as thick as the little finger, was freed; this patient (who had active pleurisy at the time of surgery) developed tuberculous empyema postoperatively, and finally bronchial fistula.

*Direct Fusion of the Pleurae.*—Three of the eight patients who had open pneumonolysis as an alternative to thoracoplasty had contralateral lesions; these were, at least at the time of operation, estimated to be quiescent. All eight patients had cavernous tuberculosis in the treated lung. Cavity diameters were approximately 2 cm. in one case, 3 cm. in four, and 4 cm. in three cases. All patients were sputum positive both when pneumothorax was induced and when pneumonolysis was performed. Each patient had been examined by thoracoscopy, and four had undergone preliminary closed pneumonolysis. Clear, serous effusion was present in two patients at operation.

Direct pleural fusion in these eight cases was, on the average, less extensive than in the A group. In three patients, however, extensive stripping was carried out; but in another three the surface stripped was no more than five cm. in diameter.

There were no deaths in this group, either in association with operation or up to the present (summer, 1950). One patient had such a severe hemothorax that evacuation of coagulate and pulmonary decortication was necessary about a week after pneumonolysis. Cavity closure was obtained in all cases but one; in this patient, ipsilateral pneumothorax was abandoned because of progression of contralateral lesions. In association with operation five patients developed persistent pleural effusion; three of these were tuberculous empyemas. Both the patients who already had an effusion at the time of surgery developed empyema postoperatively yet in one of these the area freed was not extensive. In no case was there secondary pyogenic infection of the pleural cavity.

Although pneumothorax therapy still proceeds in these eight cases, it is already manifest that in five of them pumonary function has been gravely impaired by the operation and its sequellae. For three of these patients the alternative was, however, total thoracoplasty. In the three remaining cases, such pulmonary inexpansibility, as is apparent up to the present, is very moderate.

On the basis of our present experience of open pneumonolysis in pneumothorax with direct fusion of the visceral and parietal pleurae, we may summarize thus: although this operation was employed over the period 1947-1949 on a much smaller scale than formerly, the alternative intervention—thoracoplasty—was, in our opinion, nevertheless preferable for at least two or three patients.

## DISCUSSION

The results of surgical collapse measures in pulmonary tuberculosis are often difficult to judge. Many factors are involved here, the chief being perhaps the tendency of the disease to develop fresh lesions later in its course. If the original plan of treatment cannot, in consequence hereof, be consistently adhered to, it does not follow that the treatment performed was unsuitable. As a rule, the choice of therapy is not difficult. When several methods are available, the selection may, however, be far from easy, especally as complications often appear to arise capriciously in the different interventions, and are unpredictable in the individual case.

Open pneumonolysis has regained current interest as a surgical adjunct to pneumothorax, and may be discussed along with other methods, because altered circumstances have decreased the risks associated with the operation. Hultén has, as mentioned, stressed the importance of sound aftercare; in our series, also, penicillin treatment of the pleural cavity has been valuable as prophylaxis against the mixed-infection empyema, formerly so dreaded. Empyema of this type occurred in our material only in the case where cavity rupture took place some time after operation.

It is, however, open to discussion, if the increased therapeutic resources we now command warrant that open pneumonolysis be accorded equality with, or precedence over, certain other collapse-promoting measures. During recent years, our therapeutic arsenal has gained antibiotic and chemotherapeutic preparations of great antituberculosis value; these, when surgical interventions are planned, are extremely helpful in preoperatively stabilizing and postoperatively controlling the disease. The pleural complications of open pneumonolysis can thus also be favorably affected. But despite therapeutic advances, we still await a substance which is capable of preventing and curing tuberculous empyema. When that goal is reached, the time will have come for a fresh appraisal of open pneumonolysis in competition with other collapse measures. For the present, at least, we must reckon with pleural complications in association with open pneumonolysis.

In discussing the therapeutic value of open pneumonolysis in the surgical treatment of pulmonary tuberculosis, it would appear to be most appropriate, in conformity with the cases presented above, to differentiate thus: on the one hand, open pneumonolysis as a substitute for closed division of adhesions, and on the other, as an alternative to other forms of collapse, particularly thoracoplasty.

In *short, cord-type adhesions,* closed pneumonolysis not infrequently carries manifest danger of pulmonary trauma or hemorrhage. If open pneumonolysis is performed instead, these risks are reduced or eliminated. The advantages this implies more than outweigh the risk of postoperative effusion, which hazard, in open division of cord-type adhesions, should not be greater than if the closed procedure had been employed. *As a substitute for closed pneumonolysis* in this type of case, we consider that *open pneumonolysis well merits use,* perhaps even on a greater scale than at present.

Formerly, when the effect of pneumothorax was nullified by direct fusion of the visceral and parietal pleurae, treatment was almost always discontinued in favor of other collapse measures, chiefly thoracoplasty. The idea of obtaining satisfactory collapse and conditions for continued pneumothorax by separating directly fused pleurae is attractive. On cursory contemplation, *open pneumonolysis* should be preferable to a mutilating thoracoplasty. However, the potential risks and results of both methods are of fundamental significance in the choice of surgery. There is, consequently, reason for making *a comparison of the alternatives.*

Thoracoplasty now carries comparatively small immediate risks. The atelectases which sometimes occur as early operative sequellae, clear spontaneously or respond to treatment. Pleural complications, which are chiefly associated with the opening of a free pleura, are rare. In other respects, also, the postoperative course is generally uneventful, and the ultimate results good as regards cavity healing. Some limitation of function, in degree dependent upon the scope of the thoracoplasty, naturally results, but, provided the operation is moderate in extent, diaphragmatic mobility is not affected. Not until subtotal or total thoracoplasty has been carried out is the function of the lung largely or completely lost.

In open division of directly fused pleurae, cavity perforation and hemorrhage into the pleural cavity may occur with grave immediate consequences. Moreover, tuberculous granulation tissue may be present over the entire operation field; for this reason, the possibilities of spread of the disease via lymphatic or blood vessels or by direct contact must be kept in mind. The pleurisy which occurs so often in the later course of the disease is certainly, in many cases, a sign that tuberculous lesions were opened. Pleural complications often cause lengthy invalidism and reduce the patient's strength. This applies particularly to tuberculous empyema, which occurred in about every third patient in our series. Pleural thickening with fixation of the diaphragm and pulmonary inexpansibility are the usual sequellae of pleural complications. The final result when pneumothorax is terminated is often a considerable loss of function, out of reasonable proportion to the degree of collapse necessitated by the disease, particularly the cavity, for healing.

*Comparison of the operations indicates, in our experience, that thoracoplasty contains essentially less immediate risk, and has a considerably smoother postoperative course than open pneumonolysis. As regards cavity healing, good results are obtained by both methods. In those cases of pneumothorax with direct pleural fusion, where thoracoplasty and open pneumonolysis are alternative possibilities for the attainment of the desired collapse, we believe that, with regard to risks and results, thoracoplasty should receive primary consideration. Open pneumonolysis cannot, therefore, be accorded the same recommendation for direct fusion of the pleurae as for certain types of cord adhesions.*

This view is in complete conformity with the opinion expressed by Alexander in 1937, that in the absence of contraindications, thoracoplasty is preferable as its surgical risks and postoperative complications are less than those of open pneumonolysis. We would add here, to the further advantage of thoracoplasty that, as regards impairment of function after completed therapy, the results obtained by this procedure are often substantially better than those of open pneumonolysis.

We agree with Alexander that open pneumonolysis has its justification as a method of treatment in pneumothorax with direct fusion of the pleurae, but that indications must be sharply defined. To attempt a statement of guiding principles for the employment of the operation is naturally very difficult, and opinions unavoidably diverge. Certain general aspects, however, may be worthy of observation. It is important that the patient, to whom the prospect of continued pneumothorax refills after pneumonolysis, may be misleadingly attractive, should be fully informed of the risks attached to the alternative measures; his subjective attitude may otherwise exercise a distorting influence on the choice of surgery. It should also be emphasized that thoracoscopy yields valuable information as to the extent and degree of the proposed operation, and may thus assist the choice in difficult cases. The general rule certainly is that the more extensive the direct pleural fusion, the greater the likelihood of postoperative complications. But even the freeing of very limited areas may give rise to pleurisy reaction and empyema. The risk of empyema appears to be particularly great if active pleurisy is present at the time of operation. Hedvall and Hultén also advise against open pneumonolysis in pleurisy cases.

*In the majority of patients with extensive direct fusion of visceral and parietal pleurae, it is probably advisable to refrain from open pneumonolysis and to abandon the pneumothorax.* Particularly if the type and localization of the pulmonary lesions are such that only limited thoracoplasty is required, that operation is decidedly to be preferred, not least with regard to the desirability of retaining as much as possible of the pulmonary function. Should total thoracoplasty be necessary for adequate collapse, open pneumonolysis may, with some degree of justice, be brought up in discussion. The choice of surgery will then be dependent upon the evaluation of the complications associated with the operation. The lung will sustain injury to its function irrespective of which of the two surgical methods is employed. In such borderline cases, it is the doctor's familiarity with, and estimation of the different interventions, which finally decide the method to be employed.

*Under special circumstances* which, however, have more the character of exceptional cases, *open pneumonolysis may be directly advisable,* e.g., when there is good reason to believe that the adhesions are loose, or when retention of function in the basal parts of the lung may be anticipated, as adhesion with the diaphragm need not be freed.

Even if open pneumonolysis has thus a certain range of indications, we have now accepted this as an established fact: that, in pneumothorax with direct pleural fusion, it can be contemplated for a very limited number of patients, and then only after mature deliberation.

## SUMMARY

The writers report a series of thirty-six pneumothorax patients who underwent open pneumonolysis of short, cord-type adhesions or of direct fusion of the visceral and parietal pleurae. The results justify the following evaluation of the method.

*In short, cord-type adhesions, where closed pneumonolysis implies manifest risk of pulmonary trauma or hemorrhage, open pneumonolysis is advocated.*

*In direct fusion of the visceral and parietal pleurae, the value of open pneumonolysis is considerably more limited, due to: (1) the risk of pulmonary trauma during, and hemorrhage closely following the operation, (2) pleural complications, particularly empyema, with their sequellae of pleural thickening, diaphragm fixation and pulmonary inexpansibility. Thoracoplasty, which is usually the alternative measure, is less hazardous, and as cavity closure is generally obtained by both methods, thoracoplasty is preferable in most cases. Apart from certain exceptional cases, open pneumonolysis of direct pleural fusion should only be employed when other conceivable collapse measures are so extensive that the function of the diseased lung will be greatly reduced or possibly lost.*

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# PULMONARY DECORTICATION IN TUBERCULOSIS

## A Report of 22 Cases

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PULMONARY decortication involves peeling from the surface of the pleura abnormal fibrinous layers which have formed over it.

The procedure now commonly known as decortication was first performed by Fowler[1] on Oct. 7, 1893, in the treatment of a previously drained chronic empyema of thirty-three months' duration. He removed a mass of scar tissue occupying three-fifths of the right pleural cavity from the thoracic wall, diaphragm, pericardium, and lung. The cavity was packed open with gauze and the lung expanded in twenty-eight days. However, to Delorme must go greater credit for envisioning and rationalizing the operation, although his first case did not come to surgery until January, 1894. It was this surgeon who first used the term decortication, and it is interesting to note that neither Delorme nor the surgeons who made use of the operation in the early years used the term "thickened pleura." Delorme described the pathology of the shell as false membrane encapsulating the lung. There developed very soon, however, the erroneous idea that thickened pleura was removed, and this incorrect conception of the basic pathology has persisted with few exceptions until very recently. In the early years the operation was used most frequently in combination with collapse procedures, especially the Schede thoracoplasty. A complete description of decortication appeared again in 1915 when Lilienthal described his major intercostal thoracotomy and lung mobilization. Lilienthal was the first to treat acute empyema by decortication, and developed his procedure because of dissatisfaction with the high mortality following thoracostomy. His results were quite good, although the mortality, 19 per cent, was high by present-day standards. In the 1920's and 1930's, the operation was performed relatively infrequently, and had practically fallen into disuse until 1946 when a series of papers were presented at the Annual Meeting of the American Association for Thoracic Surgery describing the experiences of American surgeons[2, 3] in the treatment of chronic organizing hemothorax by decortication. At the same meeting, Gurd[4] of Montreal presented three cases of tuberculous empyema which he had treated by decortication. His operation differed from the others in that he removed a portion of the chest wall as in a Schede procedure and the wound was packed open. Mulvihill, in discussing Gurd's[4] paper, presented a case of his own in which, through an intercostal incision, he had removed almost the entire peel encasing the lung and diaphragm in a case of unexpandable lung following pneumothorax therapy. It was felt at that time that the operation would be of little use in the treatment of a frank tuberculous empyema, and

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it was suggested that the empyema should first be sterilized before any attempt was made to decorticate the lung. However, increasingly numerous reports began to appear in the literature of the successful treatment of tuberculous empyema by this process, and it is now considered that pulmonary decortication is a most important procedure in the treatment of one of the most dreaded complications of artificial pneumothorax.

## INDICATIONS

The aim of pulmonary decortication is to remove the encasing peel that is preventing the lung from re-expanding. It is therefore indicated in the following instances:

1. Unexpandable lung following *artificial* pneumothorax therapy
   - (a) to obliterate the space,
   - (b) to increase vital capacity in order to permit surgery for the other lung,
   - (c) to replace displaced mediastinum in the presence of symptoms.
2. As an adjunct to pulmonary resection. Removal of a lobe or lung is made much easier in cases where there is marked symphysis if the plane between visceral pleura and the encasing peel is first found and decortication performed. Also, in cases of lobectomy, if rapid obliteration of the pleural space is to be obtained, as well as maximum function of the remaining lung, decortication of this lobe or lobes must be carried out before the chest is closed.
3. Tuberculous empyema.

## ALTERNATIVES TO DECORTICATIONS

*In the past, when decortication was not being used in the treatment of these* cases, the following measures were carried out:

1. *Continuous aspiration:* This, when successful, produced obliteration of the space by the marked retraction of the mediastinum to meet the chest wall which also was pulled in. This frequently produced symptoms, such as dyspnea and palpitation, and the function of the lung was not improved. Indeed, because of the necessary overexpansion of the contralateral lung, vital capacity was often decreased.

2. *Thoracostomy:* Rib resection and drainage of the chest was often necessary as an emergency, but this procedure alone rarely effected obliteration of the residual space.

3. *Thoracoplasty:* An extensive thoracoplasty was successful in obliterating the space in a considerable proportion of these cases, although when one notes the nature of the peel at operation one wonders how it ever could have been collapsed. Total thoracoplasty, of course, by obliterating the space, eliminated function of the entire lung. It was found also in some cases that while roentgenograms and aspiration indicated that the pleural space had been obliterated, and consequently the patient was discharged, yet fluid still remained to cause trouble months, and sometimes years, afterward.

4. *Operations of the Schede type:* These procedures were frequently necessary especially when thoracoplasty failed. A Schede operation involved removal

of the ribs, periosteum, intercostal bundles and pleura, completely undecking the empyema cavity and then suturing the musculo-cutaneous flap onto the visceral peel and hoping that the graft would take. Frequently, it did not and the patient was left with a large chest wall defect which took years to heal. These Schede procedures caused marked deformity, not only to the thorax but, because the lower intercostal nerves were severed, there was marked herniation of the abdomen on the side of the operation, and patients with extensive Schedes are forced to wear complicated appliances in an attempt to counteract these abnormalities.

## PATHOLOGY

It has seemed wise to discard the term "thickened pleura" to a great extent. In only a few cases is the pathology an inflammatory thickening of the visceral pleura itself. Almost always it consists of the laying down of hyalinized fibrous layers on the pleura.[1] This membrane is not intimately adherent in a large part, but is attached by a thin areolar vascularized layer which may be separated easily by blunt dissection, leaving behind *an intact and, for the most part*, fairly normal, expandable visceral pleura. This fibrous envelope may vary from a thin transparent filmlike layer to a thickness of one quarter of an inch or more. Regardless of thickness, the character of the loose areolar layer approximating the pleura is the same. In a few small areas there may be intimate attachment to the visceral pleura by tough scar, usually over areas of subjacent parenchymal disease. These areas may require sharp dissection for separation, or perhaps preferably they should be left in place. It would seem that the ease of decortication bears little relationship to the length of time the lung has been collapsed. The first patient to be presented in this series was a man who had had pneumothorax, oleothorax, and empyema for a period totalling in all thirty years. Decortication of his lung was surprisingly easy and successful.

## CONTRAINDICATIONS TO DECORTICATION

It is felt that a definite contraindication to the performance of this procedure is the presence of an open focus in the underlying lung. In these cases, if it is considered that thoracoplasty will not collapse the cavity, a combined decortication and lobectomy is done. Some surgeons feel that decortication should not be carried out in pulmonary tuberculosis for fear of reactivating an apparently quiescent focus in the underlying lobe or lung. In the limited experience of this study, reactivation of the disease in the underlying lung did not occur. It is possible that this is, in a great part, due to the pre- and postoperative use of streptomycin.

## PREOPERATIVE PREPARATION

As the procedure is elective, time is taken to make the patient's general condition as good as possible. Bronchoscopy is carried out to rule out active tracheobronchial disease, bronchostenosis (which would render expansion of the collapsed lung impossible even after decortication), and the presence of bacillary secretion from the lung to be operated on. The usual careful cardiovascular examinations and vital capacity determinations are carried out. Streptomycin, one gram daily, and procaine penicillin G, 300,000 units once daily, is begun five days before the operation. Endotracheal anesthesia is considered to be mandatory, and pentothal induction followed by cyclopropane-ether-oxygen is preferred.

### OPERATIVE TECHNIQUE

The incision is the usual posterolateral, one and the chest is usually entered between the fifth and sixth ribs after dividing them posteriorly. After cutting through the endothoracic fascia and parietal pleura, the outer parietal surface of the peel is reached. This is stripped away from the chest wall for a considerable area, and the rib spreaders are then inserted. This prior stripping of the parietal peel makes it possible to spread the ribs fairly widely apart so that an adequate exposure is obtained. The empyema cavity is entered, and an incision is then carefully made through the visceral peel until the shiny, bluish, visceral pleura comes into view. By combined blunt and sharp dissection, the visceral peel is removed entirely from the lung. The interlobar fissures are freed—a most important part of the procedure—and the parietal covering is then removed from the diaphragm, thoracic wall, and mediastinum. Areas of infolding of the surface of the lung are freed by sharp dissection. One occasionally runs across a case of true thickened pleura due to actual pleural inflammation, as in one of our patients. The visceral peel then will not strip, and, in his case, it was necessary to leave a sizeable section intact. However, there is usually enough expansion of the lung around such areas to obliterate the pleural space. Frequently, in removing the peel, the lung is damaged and at the close of the operation there are often lung air leaks. Many surgeons[1, 2, 7, 8] advise repairing these, but we feel that with proper postoperative management no harm is done by leaving them. The lung is then inflated by the anesthetist, two drainage tubes are placed, one in front at the apex and one posteriorly at the base, and the chest wall is closed in layers. Throughout the operation there is considerable oozing, and these patients require an adequate amount of blood replacement, the average being 2,000 c.c.

### POSTOPERATIVE CARE

Postoperatively oxygen is administered for four to eight hours, and the tubes are connected alternately to a continuous suction apparatus. This is a most important part of the postoperative treatment, especially if there has been any lung air leak. It is usual to connect the anterior tube for two hours and the posterior one for one hour. Streptomycin and penicillin are continued, penicillin until the lung is completely re-expanded, and streptomycin and PAS for at least fifty days.

### RESULTS

At the Nova Scotia Sanatorium, we have done one decortication for unexpandible lung, seven during the course of pulmonary resection and fourteen for

TABLE I. AGE AND SEX INCIDENCE RELATED TO INDICATION FOR OPERATION*

| AGE GROUP | UNEXPANDABLE LUNG | | WITH LUNG RESECTION | | EMPYEMA | | TOTAL |
|---|---|---|---|---|---|---|---|
| | MALE | FEMALE | MALE | FEMALE | MALE | FEMALE | |
| 10-19 | 0 | 0 | 2 | 1 | 0 | 0 | 3 |
| 20-29 | 0 | 0 | 0 | 4 | 2 | 5 | 11 |
| 30-39 | 1 | 0 | 0 | 0 | 2 | 0 | 3 |
| 40-49 | 0 | 0 | 0 | 0 | 4 | 0 | 4 |
| 50-59 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| All Ages | 1 | 0 | 2 | 5 | 9 | 5 | 22 |

*The youngest was 13 years of age, the oldest 53 years.

tuberculous empyema. Age, sex, and the indication for operation are outlined in Table I. Briefly, our results are as follows:

In the one case of unexpandable lung without empyema, the illness began with spontaneous pneumothorax nineteen months previously in a man who had had his right arm amputated for a fibrosarcoma three years before. Spontaneous collapse occurred on the left side, and a roentgenogram following it revealed a density in the right mid-lung region which eventually excavated. Decortication on the left was decided upon with the view that exploration of the right chest might be necessary. The operation was done without difficulty, and it was possible to obtain a biopsy from the surface of the lung during the procedure. The lung re-expanded uneventfully. The pathologist's report was secondary fibrosarcoma.

In seven cases, decortication was done during the course of pulmonary resection (see Table II). The presence of the peel was explained by pre-existing pneumothorax in four cases. In one, the pneumothorax had been present for less than six months; in one, nine months; and in two, twelve to twenty-four months, respectively. Two patients had pneumothorax complicated by effusion, the duration of the latter in both instances being less than six months. In one patient, the presence of the peel could not be explained as there was no history of previous pneumothorax or pleural effusion. In all cases, the spaces became obliterated promptly following operation.

It is felt that decortication is of most value in the treatment of tuberculous empyema, and, before discussing results in the fourteen cases, in which the operation was performed for this indication, it might be well to present briefly the classification of tuberculous empyema in use at the Nova Scotia Sanatorium:

Type I—serous fluid positive for tubercle bacilli, underlying lung disease controlled.

Type II—serous fluid positive for tubercle bacilli, underlying lung disease uncontrolled

Type III—frank pus positive for tubercle bacilli, underlying lung disease controlled

Type IV—frank pus positive for tubercle bacilli, underlying lung disease uncontrolled

Type V—mixed infection

Obviously, decortication is not the best procedure for types II and IV, and some cases of type V where the underlying lung is not controlled will require some other procedure.

TABLE II. DECORTICATION ACCOMPANYING LUNG RESECTION; ETIOLOGY AND DURATION OF PEEL

| ETIOLOGY | DURATION OF PEEL<br>UNDER 6 MONTHS | 6 TO 12 MONTHS | 12 TO 24 MONTHS | UNKNOWN |
|---|---|---|---|---|
| Artificial pneumothorax only | 1 | 1 | 2 | 0 |
| Artificial pneumothorax with effusion | 2 | 0 | 0 | 0 |
| No known reason | 0 | 0 | 0 | 1 |
| All reasons | 3 | 1 | 2 | 1 |

TABLE III. DECORTICATION FOR EMPYEMA-TYPE AND ETIOLOGY OF EMPYEMA

| TYPE | ARTIFICIAL PNEUMOTHORAX | PLEURISY WITH EFFUSION | POST-RESECTION OF LUNG TISSUE | TOTAL |
|---|---|---|---|---|
| I | 2 | 1 | 0 | 3 |
| III | 6 | 1 | 0 | 7 |
| V | 2 | 1 | 1 | 4 |
| All types | 10 | 3 | 1 | 14 |

The type and etiology of the empyema are outlined in Table III. Of the three type I empyemas, two complicated artificial pneumothorax and one occurred spontaneously. Of the type III empyemas, six complicated artificial pneumothorax and one was spontaneous; and of the type V, two complicated pneumothorax, one was spontaneous and one occurred following lobectomy.

The results in decortication of empyema cases are outlined in Tables IV to VII, inclusive.

The empyema was classified as type I in three of the fourteen patients (Table IV). Each of these obtained a good result, and prompt, complete, obliteration of the space took place following decortication.

In seven patients, there was a type III empyema present. Of these, four obtained an excellent result with complete re-expansion; one is classified as improved, that is, there is a small residual space present; and two are classified as poor in that the empyema cavity became larger and required further surgical measures.

In four patients, the empyema was classified as type V, that is, mixed infection with bronchopleural fistula present. Surprisingly, all four of these patients obtained an excellent result following the operation. There were no deaths in the series.

In assessing the results of treatment as related to the duration of the empyema, three patients had their empyema more than two months but less than

TABLE IV. DECORTICATION FOR EMPYEMA; RESULTS ACCORDING TO TYPE OF EMPYEMA

| TYPE | RESULT<br>GOOD* | FAIR† | POOR‡ | TOTAL |
|---|---|---|---|---|
| I | 3 | 0 | 0 | 3 |
| III | 4 | 1 | 2 | 7 |
| V | 4 | 0 | 0 | 4 |
| All types | 11 | 1 | 2 | 14 |

*Good—Complete obliteration of space.
†Fair—Small residual space.
‡Poor—Space same or larger.

TABLE V. DECORTICATION FOR EMPYEMA; RESULTS RELATED TO DURATION OF EMPYEMA

| PREOPERATIVE DURATION OF EMPYEMA | | RESULT | | | TOTAL |
|---|---|---|---|---|---|
| | | GOOD* | FAIR† | POOR‡ | |
| >2 mo.§ | <1 yr.‖ | 3 | 0 | 0 | 3 |
| >1 yr. | <2 yr. | 3 | 0 | 1 | 4 |
| >2 yr. | <5 yr. | 2 | 0 | 0 | 2 |
| >5 yr. | <10 yr. | 2 | 0 | 0 | 2 |
| >10 yr. | <21 yr. | 1 | 1 | 1 | 3 |
| All cases | | 11 | 1 | 2 | 14 |

*Good—Complete obliteration of space.
†Fair—Small residual space.
‡Poor—Space same or larger.
§> Greater than.
‖< Less than.

one year, and all three of these obtained good results (Table V). There were four in whom the duration of the empyema was more than one year but less than two years. Three of these obtained good results, and one is classified as poor. The latter is the patient in whom the operation had to be interrupted because of the deterioration of his condition on the table. Those with empyema more than two years but less than five numbered two, and both obtained an excellent result. There were two cases in which the complication had been present for more than five years but less than ten. Both spaces became obliterated completely. Three patients had empyema for more than ten years, and the maximum duration was twenty-one years. One of these obtained a good result, and in one the result was classified as fair and one as poor. In these two unsatisfactory cases, it was not possible to decorticate the diaphragm.

It would appear that the duration of the peel does not greatly affect the ultimate result.

It is of interest to relate the results obtained to the extent of the decortication, as shown in Table VI. Ten of the patients had complete decortication of the parietal and visceral peels. The entire group obtained good results. In four of the patients, the decortication was incomplete. In three of these, the dia~~phragm could not be deco~~rticated, and in one the operation had to be interrupted because of the poor condition of the patient on the table. This last named patient obviously did not obtain a good result, and two of the three patients in whom the diaphragm could not be uncovered have small to moderate residual spaces at the base of the chests, emphasizing the desirability of completely decorticating the diaphragm if a good result is to be obtained.

In the eleven empyema patients who obtained complete expansion of the lung, obliteration of the pleural space occurred in ten to twenty-six days post-

TABLE VI. DECORTICATION FOR EMPYEMA; RESULTS RELATED TO EXTENT OF DECORTICATION

| EXTENT OF OPERATION | RESULT | | | TOTAL |
|---|---|---|---|---|
| | GOOD* | FAIR† | POOR‡ | |
| Complete | 10 | 0 | 0 | 10 |
| Incomplete | 1 | 1 | 2 | 4 |
| All cases | 11 | 1 | 2 | 14 |

*Good—Complete obliteration of space.
†Fair—Small residual space.
‡Poor—Space same or larger.

TABLE VII. DECORTICATION FOR EMPYEMA; RESULTS RELATED TO EXTENT OF PARENCHYMAL DISEASE

| EXTENT OF ORIGINAL LUNG LESION | RESULT | | | TOTAL |
|---|---|---|---|---|
| | GOOD* | FAIR† | POOR‡ | |
| Minimal | 1 | 0 | 0 | 1 |
| Moderatively advanced | 5 | 1 | 1 | 7 |
| Far advanced | 5 | 0 | 1 | 6 |
| All cases | 11 | 1 | 2 | 14 |

*Good—Complete obliteration of space.
†Fair—Small residual space.
‡Poor—Space same or larger.

operatively, seven of them taking under twenty days. The tubes were not removed until it was certain that the lung was everywhere adherent.

Only one patient was decorticated without lung leak, five had slight leakage of air, and in eight there was moderate to marked tearing of lung tissue. In the last named group, three obtained a poor result, but we feel, as mentioned above, that this was due not to leakage of air which was immediately removed from the chest by continuous suction, but to the incomplete decortication.

The amount of parenchymal disease present in the lung before collapse appeared to have little effect on the eventual results of decortication (Table VII). It was noted on one or two occasions that possibly one lobe appeared fibrosed and would not expand properly, but there occurred enough overexpansion of the remaining lobe or lobes to fill the space.

No estimations as to the increase in vital capacity due to the operation were made in these cases. The results recorded in the literature[5, 6] to date disagree as to how much increased function occurs. However, in this series, the prime concern was the treatment of the tuberculous empyema, the control of which depends on the prompt obliteration of the pleural space.

One patient only had a troublesome complication. In this case, some parietal pleura was inadvertently removed with the peel, and a considerable number of lung tears also occurred resulting in extremely marked surgical emphysema. The man was quite dyspneic for about ten days, but eventually made a good recovery with complete obliteration of the empyema space.

Short case histories of our fourteen empyema cases are presented.

## CASE REPORTS

CASE 1.—D. C., white man, aged 53 years, developed pulmonary tuberculosis in 1917. Artificial pneumothorax was begun in 1918. It was maintained for twelve years and then replaced by oleothorax. The oleothorax was continued for five years. In 1938, a thoracoplasty was performed, but the space decreased only slightly in size. For the next ten years aspiration of thick pus was required three times weekly. Fluid was positive for tubercle bacilli (type V empyema). Bronchopleural fistula was demonstrated in 1948 and 1949. Pulmonary decortication was done in two stages, April 29, 1949, and May 13, 1949, with complete obliteration of the space.

CASE 2.—J. L., white man, aged 41 years, developed tuberculosis in 1926. Artificial pneumothorax was begun in 1927. A type III empyema developed during November, 1930. Aspirations were done frequently until January, 1932, when an oleothorax was substituted. The patient carried on in good health until September, 1948, when there was a recurrence of the emypema. The oil was removed and a complete thoracoplasty done from November, 1948, to January, 1949. The space was moderately decreased in size. In June, 1949, a pulmonary decortication was carried out. The diaphragm was not decorticated. The lung expanded well except at the base where a moderate-sized residual space remained. It was necessary, in March, 1950, to open this pocket widely. It was packed daily with PAS until March, 1951, when it appeared to have obliterated completely (classified as a fair result from decortication).

CASE 3.—L. C., white man, aged 24 years, developed pleurisy with effusion in January, 1948. The effusion became purulent and positive for tubercle bacilli. After several months of aspirations, a pulmonary decortication was done Oct. 7, 1949. The patient's condition became poor on the table when only the lower lobe had been decorticated and the operation had to be interrupted. The space persisted in spite of frequent aspiration, but the patient's condition was not good enough to undertake further surgery until Feb. 27, 1951, at which time two separate empyema pockets were entered and the visceral peel in these areas removed. When this was done, it was found that insufficient lung expansion was occurring. Consequently, portions of the second to the eighth ribs were removed subperiosteally. The postoperative course was extremely stormy, with marked spread to the contralateral lung. However, this disease has now almost completely resolved, and the empyema space appears to be completely obliterated (classified as a poor result from decortication).

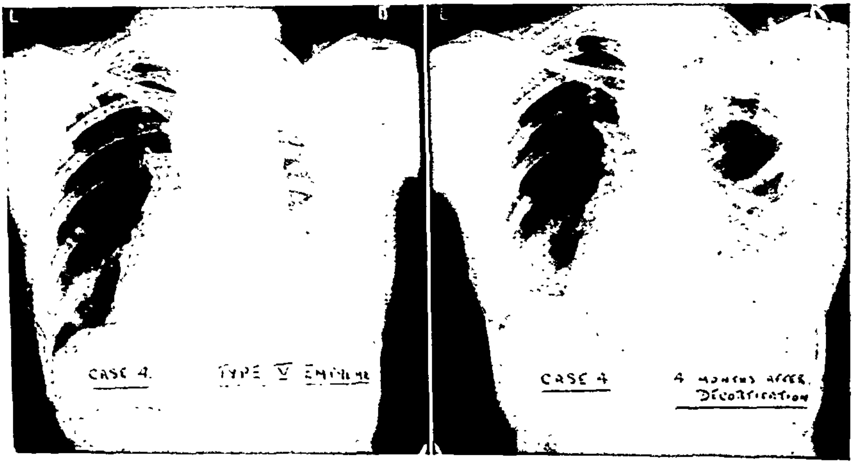

Fig. 1.—Case 4. Roentgenogram of chest before and after decortication. One of early cases requiring resection of a rib.

CASE 4.—J. R., white woman, aged 28 years, developed tuberculosis in 1946. A right artificial pneumothorax was initiated, and in January, 1947, an intrapleural pneumolysis was carried out. A massive hemopneumothorax followed and, in a matter of weeks, this became a fibrothorax. The patient's general condition improved and sputum became negative, so it was decided that she probably would not require further treatment. However, in 1948, a type V empyema with bronchopleural fistula was demonstrated. Pulmonary decortication was done on May 6, 1949, with prompt re-expansion of the lung (Fig. 1).

CASE 5.—M. T., white woman, aged 27 years, developed tuberculosis in 1948. Pneumothorax was attempted, but pleural symphysis was found. A left phrenic crush was done in December, 1948, and in March, 1949, pneumoperitoneum was begun. Atelectasis of the left upper lobe with cavity formation appeared, and in October, 1949, a left upper lobectomy was done. Following operation, a clotted hemothorax developed and was soon followed by bronchopleural fistula and the development of a type V empyema. Pulmonary decortication was done Dec. 2, 1949, with complete obliteration of the pleural space (Fig. 2).

Case 6.—N. S., white woman, aged 24 years, developed tuberculosis in 1945. A right pneumothorax was begun in August, 1946. An effusion developed some months later and became a type III empyema in February, 1948. In spite of continuous aspiration, the lung did not expand. A right pulmonary decortication was done on Jan. 27, 1950, with prompt obliteration of the pleural space. The patient remained well until October, 1950, at which time a small new infiltration was noted in the left apex. This is still present and will probably require further treatment.

Case 7.—J. M., white man, aged 44 years, developed tuberculosis in January, 1943. Left pneumothorax was begun during April, 1944, and a type I empyema developed in March, 1947. In spite of frequent aspirations, the lung did not re-expand. Pulmonary decortication was carried out on Feb. 20, 1950, with prompt obliteration of the space (Fig. 3).

Case 8.—A. H., white man, aged 31 years, developed a right-sided pleurisy with effusion during June, 1941. This became purulent and positive for tubercle bacilli in 1942. With aspirations, it was thought that the space had obliterated, and the patient remained well until September, 1948, when a type V empyema with bronchopleural fistula developed. After a period of frequent aspirations a right pulmonary decortication was done on May 17, 1950. There was complete obliteration of the empyema cavity.

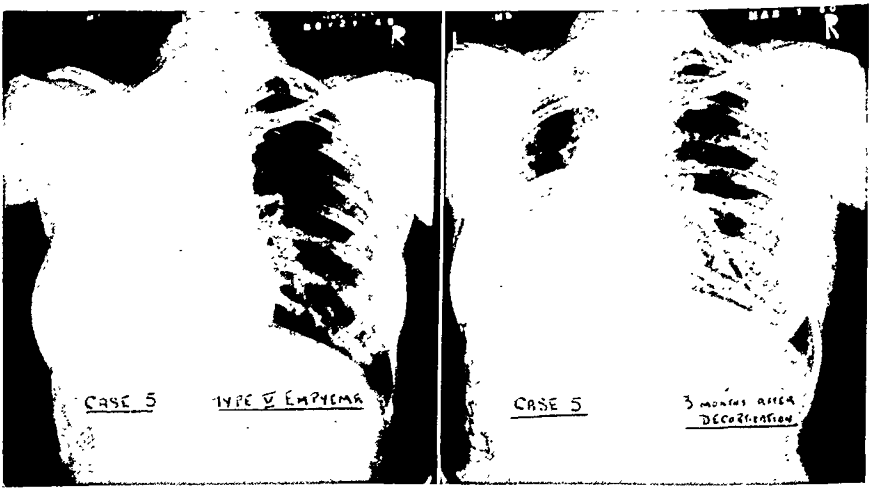

Fig. 2.—Case 5. Roentgenogram of chest before and after decortication. Also an early case for which a rib was resected.

Case 9.—H. C., white man, aged 34 years, developed tuberculosis in 1931. Artificial pneumothorax was begun promptly, and carried on for three years when the lung was allowed to re-expand. An opacity at the base was thought to be "thickened pleura." The patient remained well for fifteen years. In January, 1949, he developed a severe cough and sputum with streaking. Purulent fluid positive for tubercle bacilli was aspirated from the right chest. Continuous aspiration, phrenic crush, and pneumoperitoneum were unsuccessful in obliterating the space. Pulmonary decortication was done in April, 1950. The diaphragm could not be decorticated. Prompt re-expansion did not occur, although the space seemed to be getting smaller. In August, 1950, a marked reaccumulation of pus occurred with high fever and chills. Open drainage was carried out, and the residual space gradually decreased until now, eight months later, its capacity is about 10 c.c. (classified as a poor result from decortication).

Case 10.—M. C., white woman, aged 23 years, developed a right-sided pulmonary tuberculosis in the spring of 1949. A pneumothorax was induced March 30, 1949, and in

February, 1950, a type III empyema developed acutely. Strenuous attempts were made to expand the lung, but these failed. Pulmonary decortication was performed on May 9, 1950, and the space was obliterated in forty-eight hours (Fig. 4).

CASE 11.—J. L., white man, aged 20 years, developed tuberculosis in 1944. For the next four years his course was rather unstable, and by March, 1949, bilateral pneumothorax had been initiated. Following a right intrapleural pneumonolysis in August, 1949, a type I empyema developed. In spite of frequent aspirations, the lung proved unexpandable, and a pulmonary decortication was done on May 12, 1950. There was prompt and complete obliteration of the empyema cavity.

CASE 12.—J. B., white man, aged 40 years, developed bilateral pulmonary tuberculosis in 1941. In 1943, a left artificial pneumothorax was begun. A type III empyema developed during July, 1945, and the space was thought to have been obliterated by aspiration by October, 1948. However, in July, 1949, purulent fluid was again found in the pleural space.

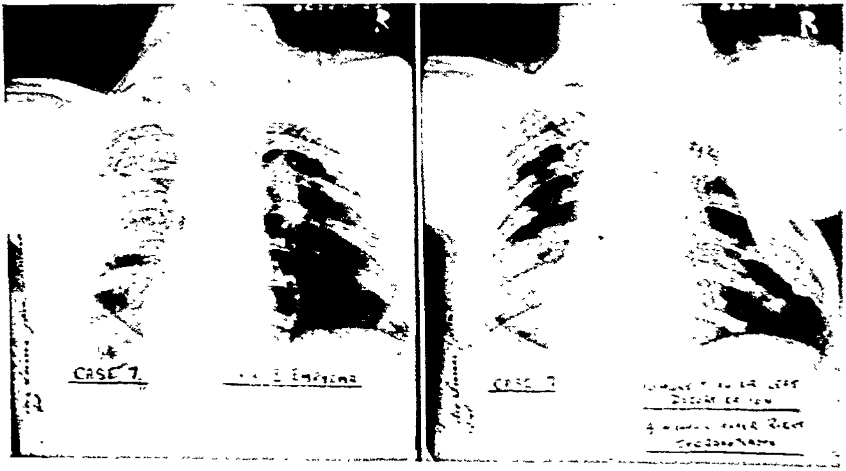

Fig. 3.—Case 7. Decortication carried out on left side to increase vital capacity and so permit thoracoplasty on right. Roentgenograms of chest before and after operations.

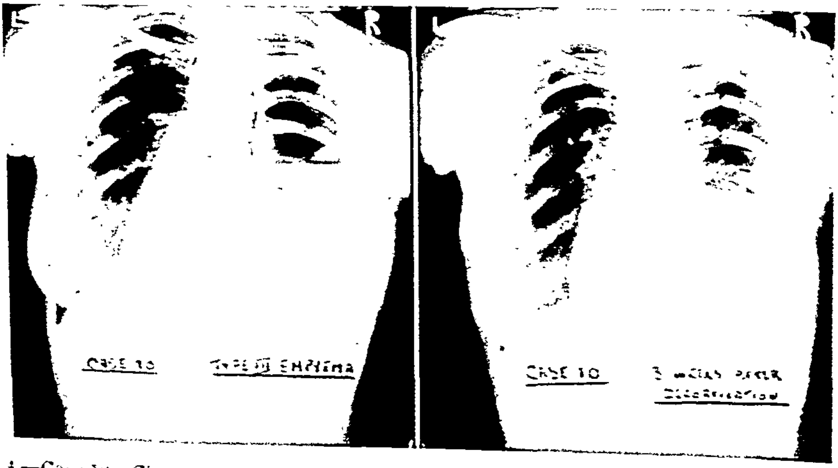

Fig. 4.—Case 10. Chest roentgenograms before and after decortication. Patient left Sanatorium four weeks after operation.

Aspirations were continued without avail. A left pulmonary decortication was done Oct. 13, 1950. The postoperative course was rather stormy because of the development of marked subcutaneous emphysema. This subsided, however, and the lung re-expanded completely.

Case 13.—R. M., white woman, aged 22 years, developed bilateral tuberculosis in 1947. Bilateral artificial pneumothorax was induced, and that on the left side was maintained for two and one-half years. On the right, a type III empyema developed in February, 1950. Sputum remained positive, and bronchiectasis was demonstrated in the right upper lobe. The empyema persisted and in October, 1950, a complete pulmonary decortication was done and the right upper lober was removed. After a period of several weeks, complete obliteration of the space occurred.

Case 14.—M. M., white man, aged 48 years, developed tuberculosis during 1946. A left-sided artificial pneumothorax was initiated in February, 1947, and this was complicated by a type I empyema during February, 1948. Repeated unsuccessful attempts were made to re-expand the lung by aspiration. A left pulmonary decortication was carried out on March 6, 1951, with rapid and complete obliteration of the pleural space.

## DISCUSSION

Although our experience is limited, we are very much impressed with the results of pulmonary decortication, especially in the treatment of tuberculous empyema. It would appear that in these cases a decortication properly and completely done will be almost 100 per cent effective in effecting a cure. It is our impression that the success of the operation will be in direct ratio to the completeness of the decortication, particularly with respect to the removal of the peel from the diaphragm. It is also very important, as far as possible, to free the interlobar fissures. While every attempt is made to treat the lung with respect, it is almost impossible to avoid a small amount of air leakage. When continuous suction is used, however, this does not appear to affect the outcome and the lung usually re-expands promptly.

The so-called unexpandable lung without empyema would seem particularly suited to this operation, as in these cases the peel is usually thin and very loosely attached and there is no infection present. In regard to resection, the availability of the decortication procedure renders unnecessary the performance of thoracoplasty or other collapse procedure to obliterate a space that could not otherwise be filled by the remaining peel-covered lobe or lobes. Pneumonectomies that were once thought impossible are now greatly simplified by the finding of the cleavage plane between the visceral peel and the visceral pleura.

## SUMMARY

1. This is a report of results with pulmonary decortication in the treatment of twenty-one cases of various pleural complications of pulmonary tuberculosis and one of unexpandable lung in pulmonary sarcoma.

2. The operation was done on one patient with unexpandable lung and no infection, on seven patients in the course of pulmonary resection, and in fourteen cases of tuberculous empyema.

3. Results were good in eleven of the fourteen empyema cases, that is, there was complete obliteration of the pleural space; one was classified as a fair re-

sult, that is, the space, while becoming smaller, did not completely obliterate; and two are classified as poor in that there was no decrease in the size of the empyema cavity without the assistance of further surgery.

4. It is considered that pulmonary decortication is the most valuable procedure available for the treatment of the aforementioned complications.

5. Brief case histories of the fourteen empyema cases are presented.

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# UNUSUAL STERNAL DEFORMITY WITH CARDIAC SYMPTOMS—OPERATIVE CORRECTION

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Baltimore, Md.

A NUMBER of structural deformities of the chest wall have been corrected by operative procedures, among them split sternum[1] and scoliosis,[2] but the thoracic deformity which is most commonly treated surgically is the deformity of the sternum known as pectus excavatum, or funnel chest. That condition is congenital, often familial, and generally progressive. There is a depression of the sternum sweeping down from the manubrium to the point of maximum depth in the lower portion of the corpus sterni. There is also an accompanying side-to-side concavity of varying depth. In most instances these patients develop a flat thin chest, a dorsal kyphos, and tend to carry their heads thrust forward. They are generally thin, puny, poorly developed individuals. In some instances, and far more commonly than is generally realized, the depressed sternum presses upon or displaces the heart, producing demonstrable physiologic disturbances. In the mildest cases there may be nothing more abnormal than electrocardiographic evidence of cardiac displacement and rotation. There may be various types of cardiac arrhythmia, there may be decrease in exercise tolerance and even cardiac failure.[3] Operation in pectus excavatum is indicated to correct the existing deformity, to prevent its progression, and to relieve symptoms which may have already occurred in those patients not operated upon in childhood.

In the present instance the sternal deformity was of an unusual type. It resembled that in pectus excavatum in that the depressed corpus sterni displaced the heart to the left, producing symptoms. However, the deformity differed from that seen in pectus excavatum. This patient was a powerfully built young man with a deep chest and good expansion. The angulation of the sternum was a double one, with an anterior bowing in the superior portion in addition to the usual concavity inferiorly. Unlike the condition in pectus excavatum, the ribs seemed to be little involved in the deformity, and there was therefore little side-to-side concavity. There was no paradoxical motion of the sternum. Operation was undertaken because of increasingly frequent attacks of what sounded like paroxysmal tachycardia.

## CASE REPORT

J. H. H., No. 463381.—The patient was an 18-year-old college student admitted for treatment of a congenital deformity of his chest. No similar deformity was known to be

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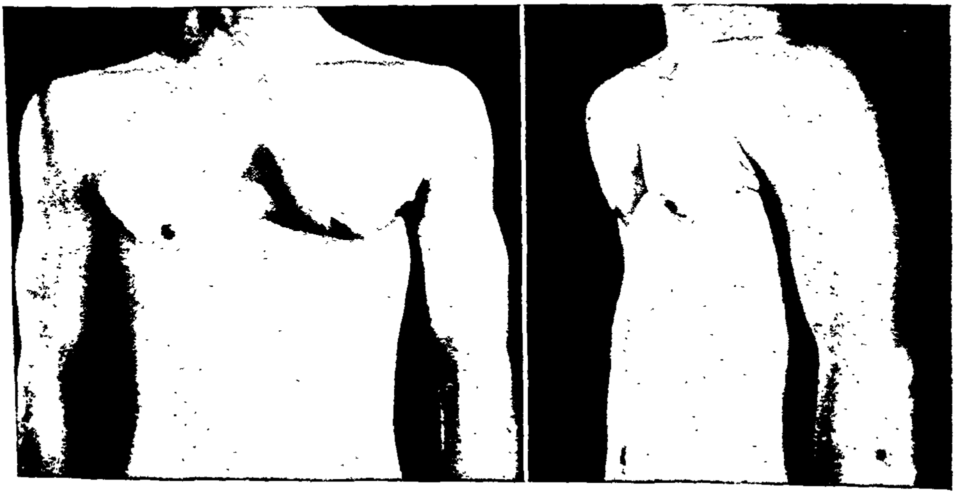
present in any member of his family. The peculiar conformation of his sternum had been noted at birth, and it had become progressively more obvious as he grew. There was no limitation in his exercise tolerance. The only symptoms which were attributable to his deformity consisted of attacks of palpitation followed by apparent slowing of the heartbeat, and heavy breathing. For about a year before admission to the hospital these attacks had occurred about once a month. He sought medical attention because of these attacks. Examination showed a powerfully built young man in excellent general health. He had a peculiar deformity of the sternum such that the manubrium stood outward from the sternomanubrial junction at an angle, thrusting forward to give him the appearance of a pouter pigeon (Fig. 1). At the junction between the manubrium and the gladiolus, the sternum angled sharply posteriorly, and again at the lower portion of the gladiolus the sternum tilted sharply forward. The deep cavity produced by the combination of these angulations served to thrust the lower portion of the body of the sternum against the heart, for roentgenograms showed a moderate displacement of the heart to the left. There was also a failure of fusion of the laminae of the second thoracic vertebra. Films taken in inspiration and expiration showed clearly that there was a definite increase in the anteroposterior diameter of the chest during inspiration, with normal descent of the diaphragm. The lateral views showed sharply the double angular deformity of the sternum with its S or Z profile (Fig. 2). The cardiac rhythm was regular and the electrocardiogram showed only a right axis deviation.



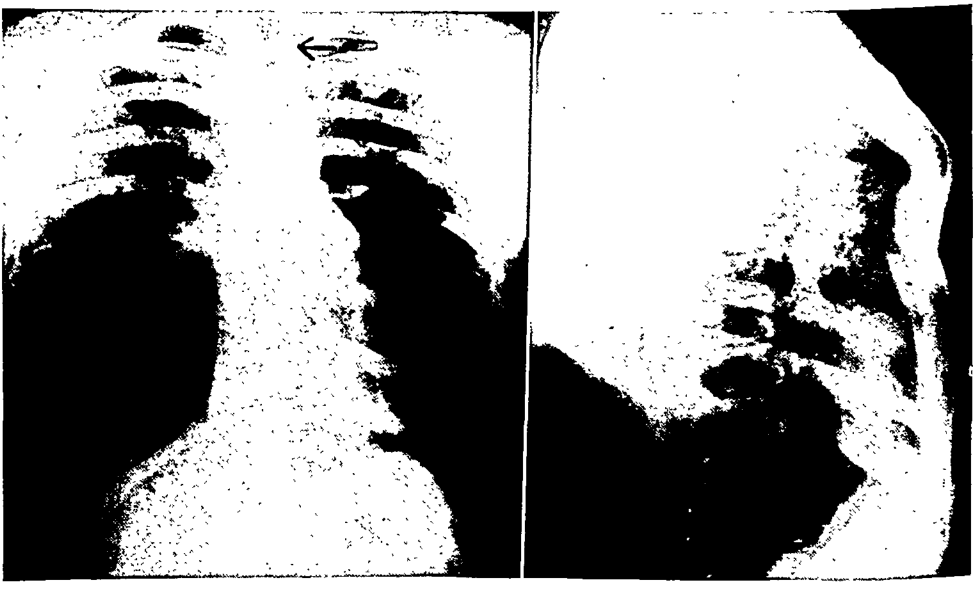
A. B.

Fig. 1.—*A* and *B*, Note the curious conformation of the chest. The manubrium slopes broadly forward to the manubriogladiolar junction where there is a sharp angulation. From this point the gladiolus inclines posteriorly to the lower third of the corpus *sterni* where the slope reverses itself and the sternum tilts anteriorly. Notice the powerful musculature and full chest, quite unlike that seen in the ordinary pectus excavatum.

Because of the history of progression of the deformity with the recent appearance of symptoms apparently attributable to it, it was felt that the symptoms could probably be relieved by correction of the deformity. This was considered not to be a typical pectus excavatum because of the sharp double angulation and because of the normal expansion of the chest without any evidence of general flatting of the chest, decrease in the anteroposterior diameter or paradoxical motion.

On June 11, 1948, under intratracheal ether anesthesia, the deformity was corrected. A midline incision was made from above the prominence of the gladiolomanubrial junction to a point well down on the epigastrium. The pectoral muscles and recti were peeled away.

The five lowermost costal cartilages on each side, which were attached to the deformed portion of the sternum, were divided, and short segments of each were resected. Inasmuch as the deformity did not extend very far laterally and the ribs were not deformed or distorted, only short segments were removed. The xiphoid was divided from the sternum and a fairly tough ligament behind it was also divided. Tough fibrous attachments between the lower portion of the sternum and the diaphragm were divided, visibly freeing the sternum. The portion of the sternum thus isolated was freed of all attachments to intercostal muscles and mediastinal tissues. The pleura was entered on the right. A transverse cuneiform osteotomy was performed at the level of greatest prominence of the convex deformity and the sternum lifted anteriorly, fracturing the posterior cortical lamella until at this point the sternum and gladiolus were level with each other. Since the lower portion of the sternum still had a concave deformity, the effect was to produce an uptilting of the lowermost portion of the sternum. This was corrected by cutting with an osteotome through most of the thickness of the sternum at the deepest point of the concavity and fracturing the lower portion of the sternum backwards, completing the realignment of the sternum. Portions of the costal cartilages were left attached to the sternum because they did not appear deformed unlike the situation characteristic of the ordinary pectus excavatum.



*A.* *B.*

Fig. 2.—Preoperative roentgenograms. *A*, Posteroanterior view. The heart is displaced to the left. The arrow points to a failure of fusion of the laminae of the second thoracic vertebra.

*B*, Lateral view. The sternal contours are accentuated by application of barium paste to the skin. The upward and forward displacement of the manubrium and the Z- or S-shaped profile of the sternum with two angulations are plainly demonstrated. In inspiration, the transverse diameter of the thorax is considerably greater than in expiration.

The cuneiform gap in the superior transverse osteotomy was closed by lifting the sternum anteriorly, and the bone was held in the corrected position at this level by three braided silk mattress sutures placed through drill holes. It was now found that the stumps of the superior most costal cartilage on either side could be sutured together with heavy silk. In order to keep the lower transverse sternal osteotomy open so as to tilt the distal segment downward and to maintain correct alignment with the body of the sternum, the bone chips removed in making the cuneiform osteotomy were packed into the open defect in the lower transverse sternal osteotomy, thus maintaining the sternum in excellent alignment (Figs. 3 and 4). The right pleura was penetrated. The patient tolerated the operation quite well although whenever the manipulation of the sternum became too forceful his blood pressure would drop from 120 mm. mercury systolic to 90 mm. mercury systolic. He received 1000 c.c. of blood in the course of the procedure.

Of great interest was the observation that when the chest was first exposed there was no room for a finger between the heart and the lower portion of the sternum which pushed directly against it, whereas at the conclusion of the operation after the division of the costal cartilages and all of the intercostal muscles on both sides and refracturing of the sternum, there was at least two fingerbreadths between the posterior border of the sternum and the heart. At the conclusion of the procedure the air which had entered the right pleural cavity was aspirated.

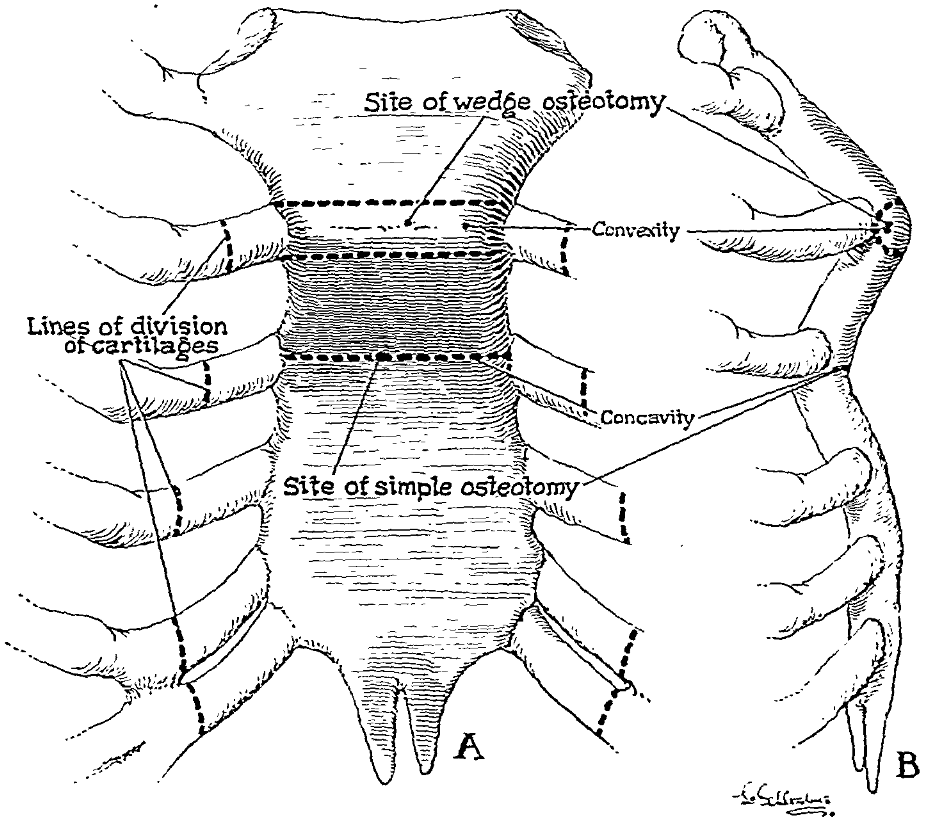


Fig. 3.—D. R. The sternum has two fairly sharp angulations. The manubrium juts out much more prominently than usual. The angle of Louis is extremely acute, and this angulation seems to emphasize the more caudal angulation which is in the reverse direction. The sternum is unusually broad, and the xiphoid is bifurcated.

The patient ran a decreasing elevation in temperature for four days after operation and accumulated a moderate amount of fluid in the right hemithorax which had to be aspirated on several occasions. There was never any evidence of paradoxical respiration. He was out of bed on the fourth day after operation, and left the hospital on the tenth day. A very satisfactory correction of the deformity was secured as evidence by his outward appearance (Fig. 5) and by the roentgenograms (Fig. 6).

He was last seen Nov. 30, 1950. He stated that since operation he had had no more of the series or runs of palpitations such as he had been having at monthly intervals before, but that occasionally he still had single momentary shocklike sensations as if his heart had missed a beat. His exercise tolerance and vigor were essentially as they were before operation and he was delighted with the cosmetic result. Examination showed maintained correction of the sternal deformity. The anterior border of the sternum was smoothly and nicely rounded. There was an inconspicuous midline scar. The sternum felt perfectly solid and

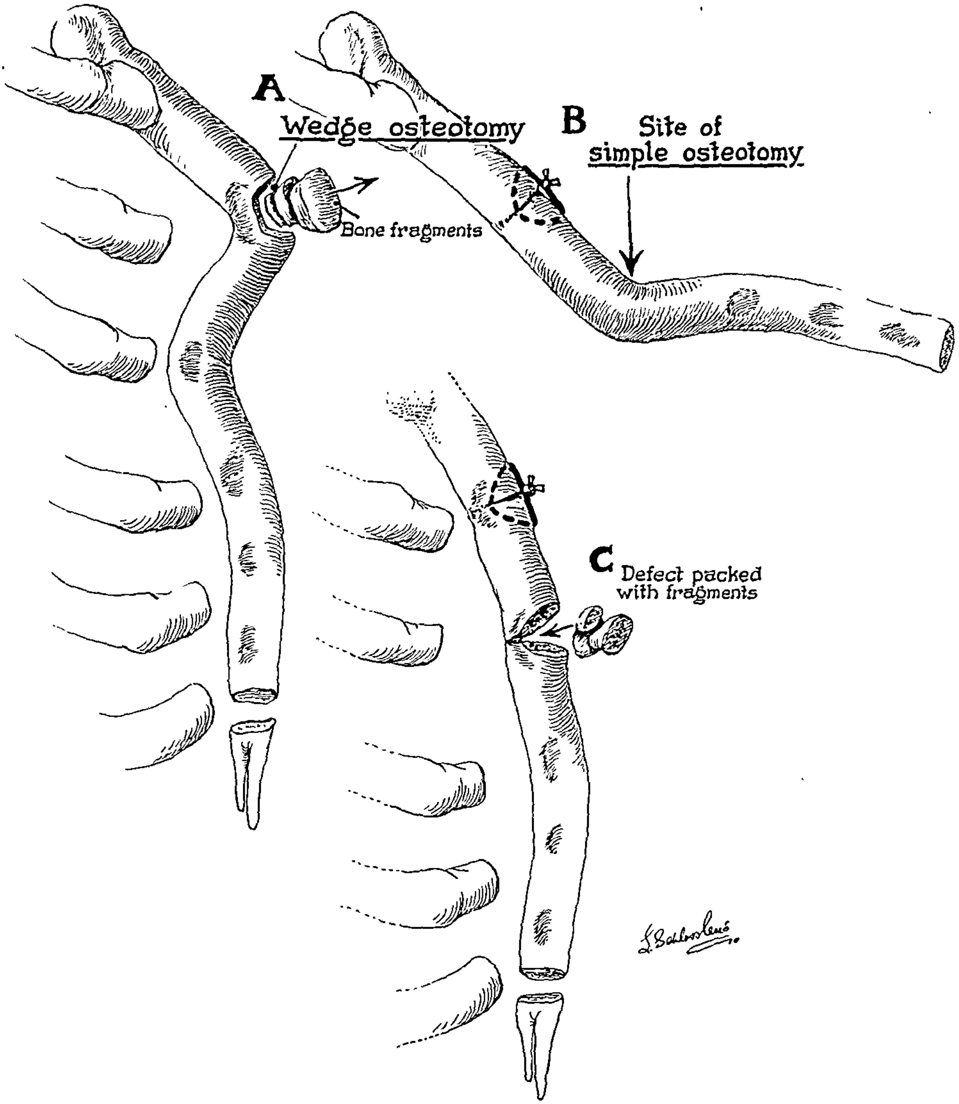


Fig. 4.—Plan of operation. After resection of segments of five costal cartilages on both sides and division of the xiphi sternal junction, two osteotomies were performed. Superiorly, a wedge osteotomy permitted the sternum to be lifted anteriorly, fracturing the posterior cortical lamella and restoring a natural alignment, as shown in *B*. This resulted in an anterior tilting of the lower portion of the corpus sterni. A simple osteotomy at the appropriate level permitted the sternum to be fractured posteriorly. The osteotomy thus opened out was packed with the fragments from the wedge osteotomy and the corrected position maintained without sutures. Superiorly, the manubrium and gladiolus were sutured together across the osteotomy with heavy braided silk. No traction or external splinting was employed.

one could not palpate any defect in the costal cartilages lateral to it. Inferior to the sternum and between the tip of the sternum and the epigastrium was a moderate sized midline defect representing the gap between the sternum and the xiphoid. The lower costal cartilages extending over from the costal margin met almost in the midline over this defect in the bony structure.

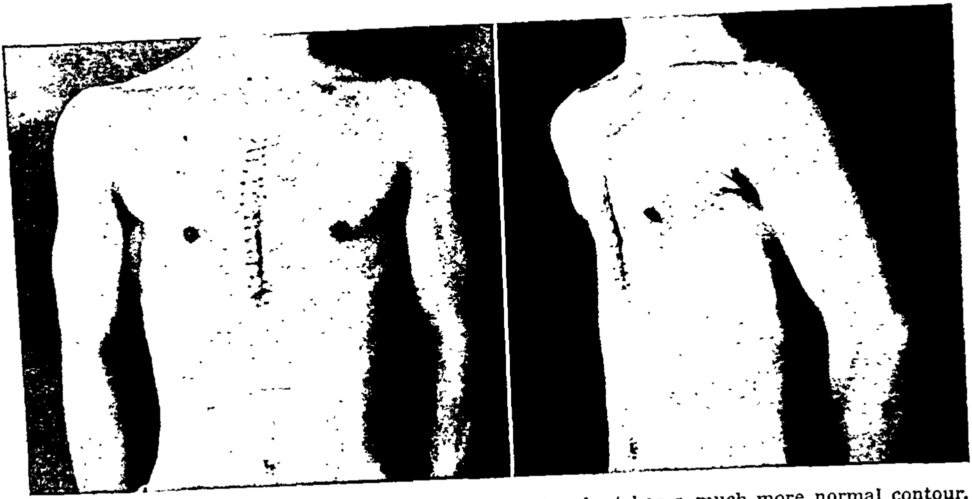


Fig. 5.—*A* and *B*, Ten days after operation. The chest has a much more normal contour, although the basic forward tilt of the manubrium remains, and, the depression of the corpus sterni having been corrected, the chest seems rather prominent. The improvement is maintained without alteration two and one-half years later.

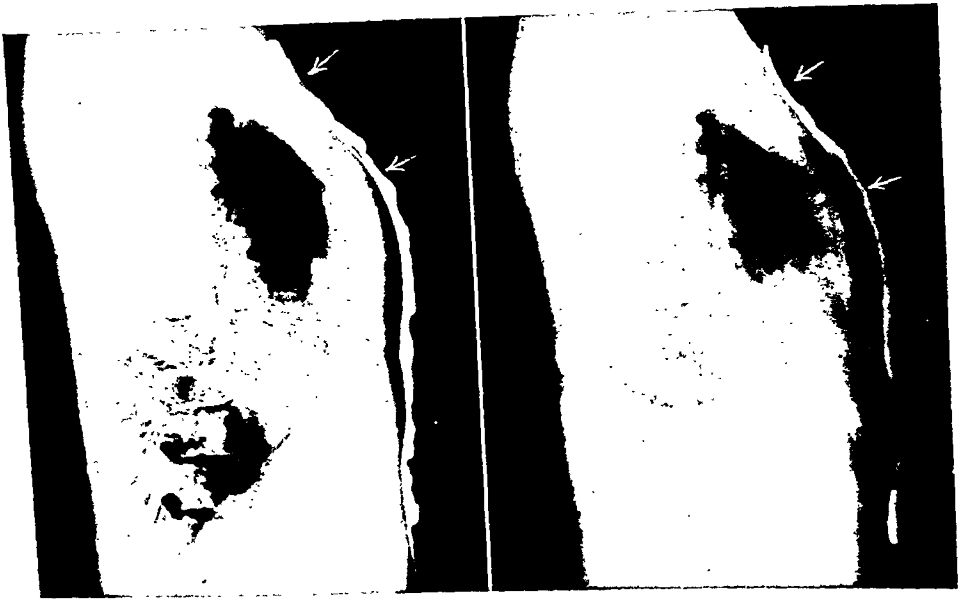


Fig. 6.—*A* and *B*, Postoperative roentgenograms. *A*, Inspiration: *B*, expiration. The chest wall is now smoothly rounded and the Z-shaped silhouette of the sternum has been corrected. The arrows point to the sites of the two osteotomies. The superior osteotomy at the manubriogladiolar junction is a wedge osteotomy, the sternum being then forcibly angulated anteriorly, fracturing the posterior cortical lamella. The lower osteotomy was spread open by fracturing the distal sternum posteriorly. Bone chips from the first osteotomy were placed in the second as wedges to hold it open. There is good expansion of the chest.

## COMMENT

In this instance an atypical funnel chest appeared to produce cardiac symptoms. The operation differed from that usually employed[4] in that two transverse osteotomies were required; very short segments of five costal cartilages on each side were resected and the sternal ends of all of them were left long. It was actually possible to resuture the second costal cartilages to the sternal ends. The correction secured was entirely satisfactory, and the symptoms appear to have been relieved. An operation of this magnitude in a powerfully built adult is a considerable procedure. There is every reason to undertake such procedures in childhood when structures are smaller and more readily handled and the deformity less pronounced.

From the technical standpoint it is of interest that even in this heavily muscled man with an unusually thick broad heavy sternum, no external traction was required and silk sutures alone securely maintained the sternum in corrected position. Three years later the chest is solid, and the achieved correction has been maintained.

## SUMMARY

A case is reported of a curious and atypical sternal deformity in an 18-year-old boy who had increasingly frequent attacks of paraxysmal tachycardia apparently due to pressure upon the heart and displacement of it.

Resection of small segments of five costal cartilages on each side, division of the xiphi sternal joint, and transverse osteotomies of the sternum at each of the levels of angulation corrected the deformity and relieved the symptoms.

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# EPIPHRENIC DIVERTICULA OF THE ESOPHAGUS

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EPIPHRENIC diverticulum of the esophagus is a rarity. After exhaustive study of all the medical literature, 126 reported cases were found. These were the cases that were sufficiently described to be reliable enough to be included. Many reported cases were found not to be true epiphrenic pulsion diverticulum, and therefore were not included in this report. A distinction must be made between true epiphrenic diverticula and traction, or functional diverticula that have other significance. An epiphrenic diverticulum is one that is located just above the diaphragm, and constitutes a pulsion or pressure diverticulum. The diverticulum will contain all the layers of the esophagus; the weakness, however, being most likely in the muscular layer of the esophagus. There are several theories as to why the muscular layer is weak. It may be congenitally weak or weakened by a localized esophagitis, and the constant pressure of the food upon a congenitally weak portion causing a diverticulum. This constant pressure may be greatly increased due to an atresia of the lower end of the esophagus or a neurogenic cardiospasm so that a normal musculature may be weakened. There may be destructive degenerative changes of intramural ganglia in the region of the esophagus that may weaken the muscular layer. Another interesting fact is that about 65 per cent of the cases of epiphrenic diverticulum have an associated cardiospasm. This might be due to similar neuromuscular defects or it may be secondary, due to reflex action originating in the inflamed wall of the diverticulum of the esophagus.

The first reported cases were those found in autopsy material. With the advent of the roentgen ray, the diagnosis could definitely be made more frequently. Symptoms of the diverticula of the esophagus were usually not very clear cut or directly related to the esophagus. Many of the cases reported were incidental findings, although some of the patients had one or more symptoms referable to the gastrointestinal tract leading to roentgenographic studies. Those patients who had symptoms directly referable to the esophagus had one or more of the following complaints: dysphagia, epigastric pain or soreness, vomiting or regurgitation, substernal distress, gas on the stomach, anorexia, belching, bloating, loss of weight, cough, bad breath, gurgling noises in the sac, heartburn, nausea, regurgitation of blood, and hiccough. The severity of the symptoms varies from very slight to severe enough to cause inanition and even emergency operation.

Depending on the severity of symptoms, the treatment varied. Many of the reported cases had no therapy at all; others had no more than bland diets,

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TABLE I. ALL RECORDED CASES OF EPIPHRENIC DIVERTICULA AND THE METHOD OF TREATMENT

| AUTHOR | YEAR | NO. OF CASES | TREATMENT | | RESULT | REFERENCE | COMMENT |
|---|---|---|---|---|---|---|---|
| | | | CONSERVATIVE | OPERATIVE | | | |
| Hausman | | 1 | 1 | | | 14 | |
| Stark | | 1 | 1 | | | 14 | |
| Tenenes | | 1 | 1 | | | 14 | |
| Ockonimedes | 1882 | 1 | 1 | | | 14 | |
| Reitzenstein | 1898 | 1 | 1 | | | 14 | |
| Borsche | 1900 | 1 | 1 | | | 14 | |
| Jung | 1900 | 1 | 1 | | | 14 | |
| Zweig | 1901 | 1 | 1 | | | 14 | |
| Kaufman | 1909 | 1 | 1 | | | 14 | |
| Keinbock | | 1 | 1 | | | 14 | |
| Steirlein | 1916 | 2 | 1 | 1 | Died | 10 | Transpleural excision of sac. Died of leakage of suture line |
| Enderlen | 1910 | 1 | | 1 | Died | 10 | Extrapleural excision of sac. Died of cardiac failure |
| Carman | 1920 | 1 | 1 | | | 14 | |
| Freund | 1921 | 3 | 3 | | | 14 | |
| Dachter | 1921 | 1 | 1 | | | 14 | |
| Clairmot | 1921 | 1 | | 1 | Successful | 32 | Resection extrapleurally |
| Bensomde | 1922 | 2 | 2 | | | 14 | |
| Haenioche | 1923 | 1 | 1 | | | 14 | |
| Dessecker | 1923 | 1 | 1 | | | 10 | |
| Kramer | 1924 | 1 | 1 | | | 14 | |
| Henschen | 1924 | 2 | | 2 | 1. Successful<br>2. Successful | 19 | 1. Diverticulo-gastrostomy (intrathoracic)<br>2. Fistula made between stomach and diverticulum (abdominal approach) |
| Horst | 1925 | 2 | 2 | | | 14 | |
| Vogel | 1925 | 1 | 1 | | | 14 | |
| Guertz | 1925 | 1 | 1 | | | 14 | |
| Nicon | 1925 | 2 | 2 | | 1 Died<br>1 Fair | | |
| Sauerbruch | 1927 | 1 | | 1 | Successful | 26 | Excision of sac |
| Moynihan | 1927 | 2 | 2 | | | 32 | Dilation of cardiac end of esophagus with bougies in 1 case and through thoracic gastrostomy in 1 case |
| Smith, L. A. | 1928 | 2 | 2 | | | 14 | |
| Wright | 1928 | 1 | 1 | | Fair | 47 | |
| Burgess | 1928 | 1 | | 1 | Successful | 43 | Intra-abdominally dilated cardiac through stomach, pulled esophagus down, excised diverticulum and anastomosed esophagus to stomach |

TABLE I—CONT'D

| AUTHOR | YEAR | NO. OF CASES | TREATMENT |  | RESULT | REFERENCE | COMMENT |
|---|---|---|---|---|---|---|---|
|  |  |  | CONSERVATIVE | OPERATIVE |  |  |  |
| Raoust | 1928 | 1 | 1 |  | Poor | 36 |  |
| Sessior | 1928 | 1 | 1 |  |  | 14 |  |
| Fitzgibbons | 1929 | 1 | 1 |  |  | 14 |  |
| Gally and Bernard | 1929 | 1 | 1 |  | Good | 14 | Dilation of cardiospasm with bougies |
| Fick | 1929 | 1 |  | 1 | Successful | 12 | Transpleural approval, removal of sac, section of phrenic nerve and fashioning of pedicle flap of diaphragm which was sutured over esophagus wound |
| Tiprez | 1930 | 1 | 1 |  |  | 44 |  |
| Quartero | 1931 | 1 |  | 1 | Fair | 36 | Transpleural resection. Slight insufficiency of sutures and leak which closed |
| Parcelier | 1932 | 1 |  | 1 | Died | 34 | Transpleural resection. Died of erosion into aorta |
| Adam | 1933 | 1 |  | 1 |  | 14 | Abdominal approach for diverticulectomy |
| Granet, E. | 1933 | 1 | 1 | 1 |  | 14 |  |
| Raven | 1933 | 1 | 1 |  |  | 39 |  |
| Barrett | 1933 | 1 |  | 1 |  | 4 | Resection extrapleurally |
| Vinson, P. P. | 1934 | 24 | Medical (All) |  | Variable | 46 | Two double diverticula. One multiple diverticula |
| Lederer, A. | 1934 | 1 |  | 1 |  | 25 |  |
| Kosenork, B. D. | 1935 | 1 | 1 |  |  | 33 |  |
| Hautefeuille | 1936 | 1 | 1 |  |  | 17 |  |
| Chevallier | 1936 | 1 | 1 |  | Fair | 7 |  |
| Neuber | 1938 | 3 | 2 | 1 | Fair | 33 | Transthoracic diverticular resection. Fistula for 3 months, requiring thoracoplasty for closure |
| Lelong | 1938 | 1 | 1 |  | Good | 27 | Infant |
| Milanes | 1939 | 1 | 1 |  |  | 30 |  |
| Harrington | 1940 | 4 |  | 4 | Successful | 16 | Resection transpleurally |
| Holinger | 1940 | 1 | 1 |  | Fair | 20 | Double diverticula in lower one-third of esophagus. (Dilatation of esophagus) |
| Chebotarev | 1940 | 4 | 4 |  |  | 7 |  |
| deAmesti | 1944 | 1 |  | 1 | Successful | 9 | Transpleural excision |
| Udando | 1944 | 1 | 1 |  |  | 45 |  |
| Abbott | 1945 | 1 |  | 1 | Died | 1 | Esophago-gastrostomy. Transpleural complicated by empyema and pericardia esophageal fistula |
| Janes | 1946 | 4 |  | 4 | Successful | 21 | Resection transpleurally |

TABLE I—CONT'D

| AUTHOR | YEAR | NO. OF CASES | TREATMENT |  | RESULT | REFERENCE | COMMENT |
|---|---|---|---|---|---|---|---|
|  |  |  | CONSERVATIVE | OPERATIVE |  |  |  |
| Ramos | 1947 | 1 | 1 |  |  | 37 |  |
| Schena | 1947 | 1 |  | 1 |  | 42 | Resection extrapleurally |
| McNealy and Glassman | 1947 | 1 |  | 1 | Successful | 29 | Intra-abdominal esophagogastrostomy |
| Kay, E. B. | 1947 | 2 | 1 | 1 | Successful | 23 | Resection extrapleurally |
| Forrester-Ward, M. N. | 1948 | 1 |  | 1 | Successful | 13 | Resection extrapleurally |
| Monod | 1948 | 1 |  | 1 | Successful | 31 | Excision of sac complicated by lung abscess most likely caused by diverticulum |
| Smelt | 1948 | 1 |  | 1 | Successful | 43 | Transthoracic excision |
| Lahey | 1948 | 8 |  | 8 | Successful | 24<br>2 | (1) Suture of sac upward dependent drainage<br>(2) Resection of sac transpleurally |
| Poppe and Berg | 1949 | 1 |  | 1 | Successful | 35 | Transthoracic esophagogastrostomy |
| Harrington | 1949 | 4 |  | 4 | Successful | 16 | 3 Diverticulectomy. 1 Invagination of small diverticulum with plication of dilated walls of esophagus |
| Johnstone | 1949 | 3 | 3 |  |  | 22 |  |
| D'Abreu | 1949 | 1 |  | 1 | Successful | 8 | Congenital type in 6-year-old girl. Diverticulectomy and lobectomy for concomitant bronchiectasis |

sedatives, and antispasmodics. Other types of palliative therapy consisted of sporadic lavaging of the sac or dilation of the cardiac end of the esophagus with hydrostatic dilators or sounds. More definitive therapy was gastrostomy, but that was done only when the sac becomes so large that it would not permit normal alimentation or when esophagitis in and around the sac did not allow oral feedings.

Of the 126 reported cases, forty-three, or 33 per cent, were operated upon for treatment of the epiphrenic diverticulum. Of these forty-three cases, there were only four operative deaths, a mortality of 9 per cent. However, the use of surgery to correct the symptoms of epiphrenic diverticula of the esophagus has only recently been generally adopted. The reasons for this late start in surgical treatment was the fear of the earlier surgeons of intrathoracic surgery. Entering the pleural cavity was not safely accomplished until the methods of controlled anesthesia, especially the development and improvement of endotracheal anesthesia, the improved methods of blood transfusion and resuscitation, and the use of chemotherapy and antibiotic therapy were in general use. The other barrier to esophageal surgery was the lack of a peritoneal covering to the esophagus, thereby making for a more precarious suture line. However, with careful technique and gentle handling of the tissues and care in maintaining the blood supply of the esophagus, this handicap has for the most part been overcome.

The operation of choice is one which would eradicate the pathologic lesion, the diverticulum, and the obstruction or spasm at the cardiac end of the esophagus. There have been many operations done, indicating that one type of operation will not cover all types of pathology. Among the operations performed were: extrapleural resection of sac (Enderlen and Fick), diverticulogastrostomy, abdominally (Lotheissen) or by the transthoracic approach (Henschen and Poppe), invagination of small diverticulum (Girard), suspension of the diverticulum for dependent drainage (Lahey and Holinger), and transpleural resection of sac and direct suture (Quartero, Harrington, Janes, etc.).

The *indications for operation* are:

1. A diverticulum, which because of its size interferes with deglutition or which because of stasis has infected retained food within, causing various gastrointestinal symptoms.
2. A diverticulum which is associated with referred pain due to pressure on the vagus or sympathetic nerves.
3. A diverticulum which is associated with cardiospasm or associated with atresia of the esophagus, congenital or acquired, which would interfere with proper nutrition.
4. A diverticulum which has become ulcerated causing localized peptic esophagitis.
5. A diverticulum that may undergo malignant changes.

It is agreed that the best approach to the esophagus is the transthoracic one, and that the extrapleural approach should no longer be used. The abdominal route is almost always an inadequate method for complete exposure, and is not now frequently used. With the perfection of fine silk technique and meticulous repair, the Lahey suspension operation has been discarded for the complete operation of diverticulectomy. The operation that has been most adequate to date has been the resection of the sac over clamps and the careful closure of the esophageal defect. This is all done in one stage, inasmuch as the location within the pleural cavity does not allow exteriorization for a two-stage procedure. In difficult cases, or when the diverticulum was too large or too close to the diaphragm, an anastomosis was done between the diverticulum and the stomach. This has proved fairly satisfactory, but it is not a complete operation.

The method of treating the diverticula and the atresia of the esophagus presented here has not heretofore been reported in the literature. This method consists of resection of the distal portion of esophagus containing the diverticula, the area of atresia, and anastomosing the esophagus to the stomach which is brought up into the chest. This allows for the complete eradication of the lesion and for the use of the gastric peritoneal layer to reinforce the suture line of the anastomosis.

## CASE REPORT

J. M. (Flower and Fifth Avenue Hospital, No. 5957), a white woman, 54 years old, was admitted on July 3, 1947, because of increasing difficulty in swallowing.

*History:* For the past year, the patient noticed increasing difficulty in swallowing. This condition progressed so far that on admission she could swallow only water. Most solid foods, excepting farina, could not be swallowed. Her weight dropped from 143 pounds to 98 pounds. Seven years previously, the patient had developed a generalized osteoarthritis, progressively involving practically all joints. This osteoarthritis compelled her to become a semi-invalid. For the past few months, there had been very little pain from her arthritis. Past history was noncontributory.

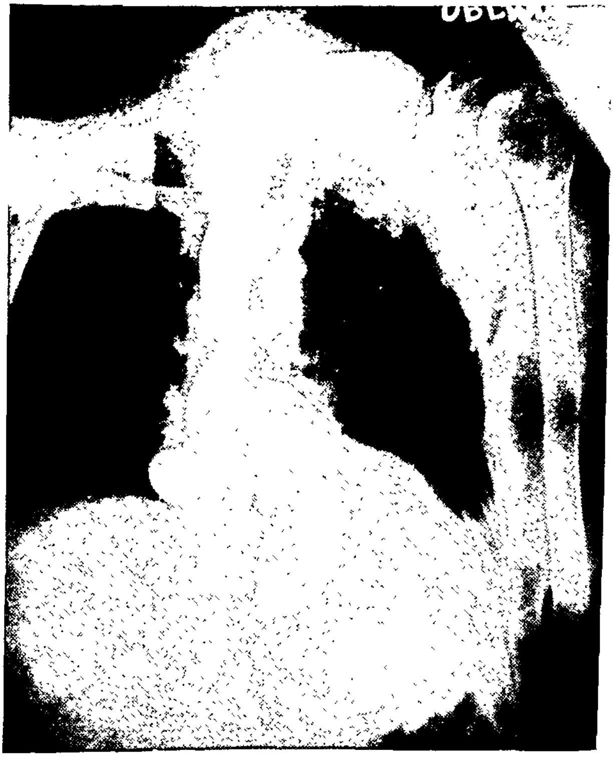


Fig. 1.—Case 1. Radiograph showing double diverticulum at lower end of esophagus and associated constriction.

*Physical Examination:* The patient presented the picture of a generalized arthritis enforcing confinement to bed. There was a pronounced loss of weight and a marked neuromuscular atrophy. Blood pressure was 138/82. Her general examination was normal.

*Laboratory Data:* Urine was normal except for one plus acetone. Blood examination showed an average normal cell count. Blood Wassermann was negative.

*Roentgenographic Examination:* Roentgenograms of the esophagus and stomach taken on June 24, 1947, showed the diameter of the esophagus down to the ninth rib posteriorly to be normal. There were two diverticula extending to the right, and posteriorly, just above the cardia. The cardiac canal and fundus of the stomach were within normal limits (Fig. 1).

*Operation:* On July 8, 1947, after suitable preparation by intravenous fluids, vitamins, blood transfusion, lavage of stomach and esophagus, and intramuscular penicillin, the patient was operated upon, under intratracheal cyclopropane, ether, and oxygen anesthesia. An incision was made over the eighth intercostal space from the angle of the eighth rib and extending downward over the cartilage of the eighth rib on to the abdominal wall, and then parallel to the outer border of the left rectus muscle to the level of the umbilicus. The peritoneal and pleural cavities were then entered, and the diaphragm was split radially from the costal cartilage to the esophagus, converting the abdominal and pleural cavities into one

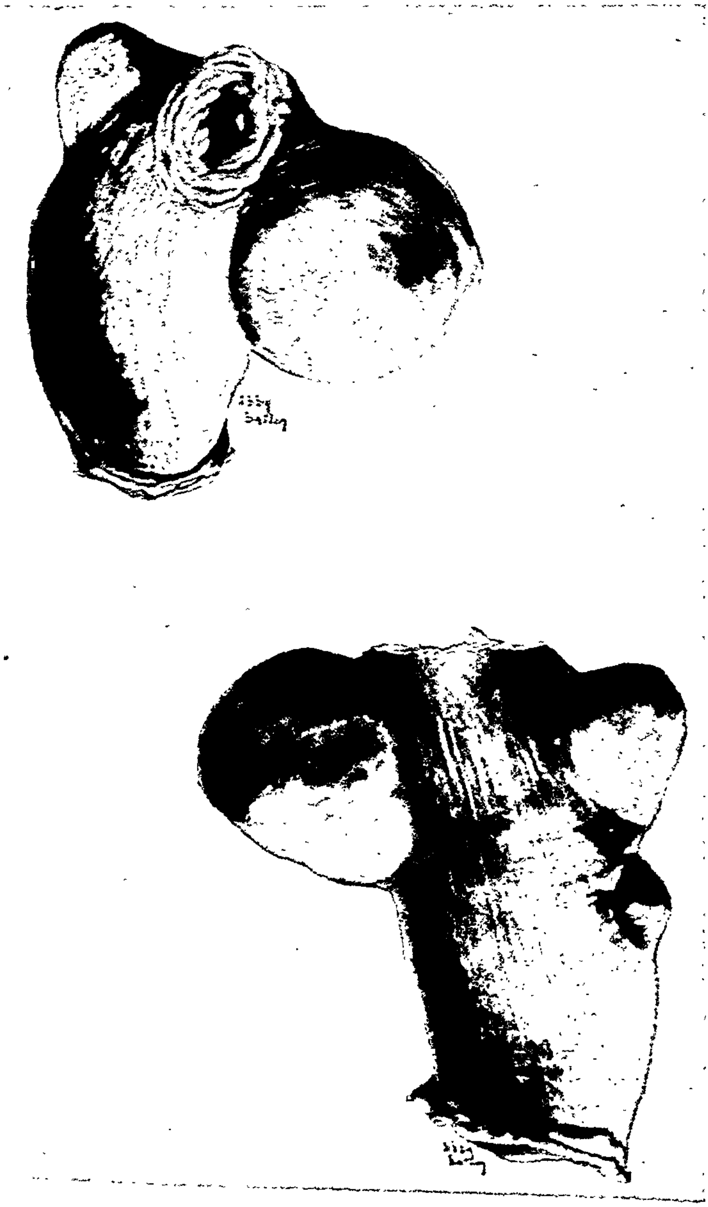


Fig. 2.—Case 1. Drawing of exterior of operative specimen of resected esophagus and diverticula.

large exposure. The left lung was densely adherent to the parietal pleura and to the diaphragm, and this was dissected off. The pleura over the esophagus was then incised from the arch of the aorta down to the diaphragm. The esophagus with the diverticula was then lifted from its bed with necessary hemostasis, care being exercised to avoid injury to the right pleura where the diverticula were adherent. The stomach was then mobilized by first tying off the left gastric vessel near the celiac axis and then separating the attachment of the stomach from the spleen and transverse colon, by ligation of the left gastroepiploic and the vas brevia, with preservation of the epiploic arch, finally ligating the right gastric and the right gastroepiploic arteries. This made it possible to draw the stomach through the split diaphragm into the chest without any degree of tension. The blood supply of the stomach seemed adequate. The distal end of the esophagus was then resected with a portion of the cardia of the stomach and the opening then closed with two layers of interrupted fine silk. The proximal end of the esophagus was resected about two inches below the arch of the aorta, and well above the diverticula. A 2 cm. oval opening was made in the anterior wall of the stomach, and a terminolateral anastomosis was then effected between the end of the esophagus and the oval opening in the anterior wall of the stomach, using fine interrupted silk sutures in two layers. The stomach was then secured to the parietal pleura to relieve tension on the suture line. The diaphragm after careful suture to the fundus of the stomach was then reconstructed with interrupted silk sutures. Two hundred thousand units of aqueous penicillin were left in the chest, and a catheter was placed through the tenth intercostal space in the midaxillary line for underwater drainage. The chest and the abdominal incision were closed in layers with chromic catgut. Interrupted silk sutures were used for the skin. The patient was given 1000 c.c. of whole blood during the operation, and she withstood the procedure well.

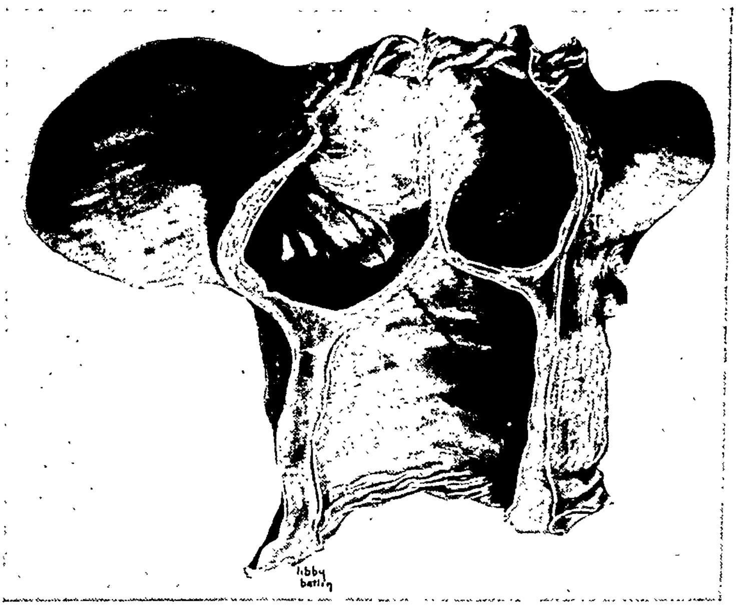


Fig. 3.—Case 1. Drawing of interior of resected esophagus showing probe through lumen of esophagus at the site of septum below diverticula.

*Course:* The patient had surprisingly little pain and temperature, considering the extensive surgical exposure. There was no dyspnea at any time. After the third day, the patient was allowed to swallow some clear fluid, deglutition being uneventful. Increasing amounts of food were gradually allowed, until by the seventh postoperative day the patient was taking a regular diet. She was discharged on the twelfth postoperative day.

The patient has been followed since and has remained well except for continuation of her arthritis. She gained 27 pounds in the first three months after operation. A postoperative gastrointestinal series showed a normal functioning esophagogastrostomy with resection of the lower end of the esophagus, and the fundus of the stomach lying above the diaphragm (Fig. 4).

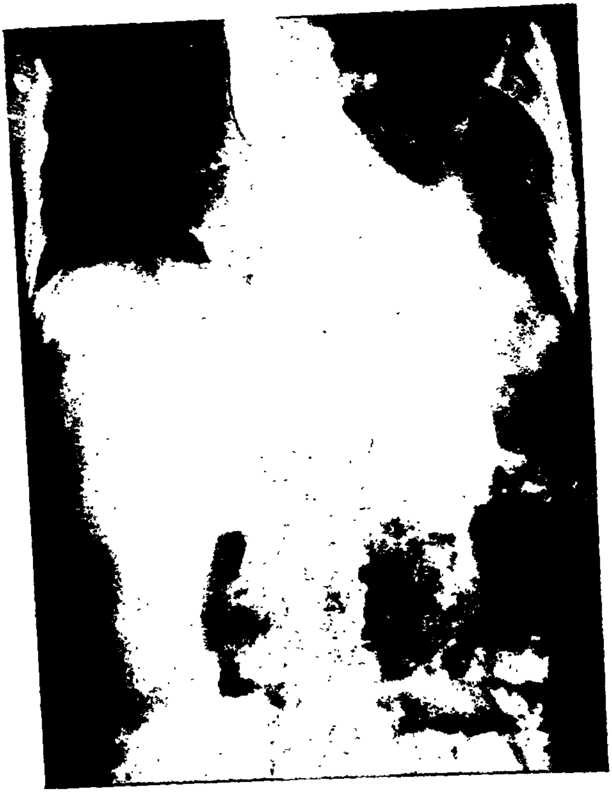


Fig. 4.—Case 1. Radiograph showing postoperative esophageal gastrostomy at lower third of the esophagus with resection of the lower end of the esophagus, and fundus of the stomach above the diaphragm.

*Pathologic Report:* The specimen consisted of the lower portion of an esophagus, measuring 6 by 3.5 cm., manifested in a tubelike structure, the cephalad portion of which contained two lateral pouches measuring 4 by 3.5 by 2.5 cm. and 2 by 2 by 2 cm. The openings into these pouches measured 2 by 1.5 cm. in their greatest diameters. The length of the esophagus joining the cardiac end of the stomach extending below the diverticula measured 3.5 cm. On section the inner lining was smooth. A short distance below the ostia of the diverticula there was a septal-like structure traversing the lumen of the esophagus occluding the lumen, except for a small opening measuring 0.5 cm. in diameter. The pathologic diagnosis was diverticula of the esophagus and partial atresia of distal end of the esophagus. (Figs. 2 and 3.)

*Comment:* From the description of the gross pathology and the study of the illustration (Fig. 3), the mechanics of the formation of these diverticula are revealed. The lumen of the esophagus was closed by a diaphragm except for an opening about 0.5 cm. wide. Without any previous history of injury or the swallowing of an escharotic, it is difficult to explain the etiology of the stricture. With the repeated attempts at swallowing, the intraesophageal pressure increased, becoming greatest just above the stricture. The weak areas in the esophageal wall finally developed, permitting the herniation of mucosa through a muscle hiatus. These diverticula should therefore be considered of the pulsion type.

CASE 2.—Mr. J. B. (Flower and Fifth Avenue Hospital, No. 4503), aged 49 years, entered the hospital on May 23, 1948, with the chief complaint of choking on food, progressively increasing for the past one and one-half years. The patient began having difficulty in swallowing dry food. He had a feeling of tightness in the throat. The episodes had increased in frequency. No vomiting was present, and there was no excessive weight loss.

*Physical Examination* was essentially normal except for a blood presure of 180/108.

*Laboratory:* Urine normal, hemoglobin 102 per cent, red blood corpuscles 6.2 million, white blood corpuscles 10,100, with a normal differential.

*Roentgenograms* taken May 11, 1948, before entering the hospital, showed a normal chest with normal heart shadow.

The esophagus, as seen fluoroscopically, on routine films, and on spot films, showed the presence of a diverticulum arising in the lower third of the esophagus. It arose from the anterolateral wall. The diverticulum sac measured approximately an inch in diameter. The entrance to the diverticulum was large, and it did not retain barium after the evacuation of the esophagus. The stomach showed no evidence of intrinsic defect.

*Course:* Because of the increasing symptoms, it was decided to operate upon the patient and remove the diverticulum.

*Operation:* Under a combination of intratracheal anesthesia with cyclopropane and curare, the chest was opened. The incision was made through the seventh intercostal space, and extended upward along the lateral border of the vertebral column. The downward extension of the incision was through the costal cartilage on to the abdominal wall. The chest was entered through the intercostal incision after sectioning the sixth and seventh ribs at their angles. The pleura overlying the esophagus was incised, and the esophagus with its diverticulum mobilized from its bed. The diverticulum was situated about two inches below the hilum of the lung and projected to the left. It had a broad base measuring about one inch in diameter, with the diverticulum itself measuring a little more than 1½ inches in diameter. It was globular in shape, and apparently had all the coats of the esophagus. The esophagus was mobilized down to the esophageal hiatus. The diaphragm was then incised and the abdominal cavity entered. The proximal half of the stomach was mobilized by ligating the left gastric vessels and gastroepiploic arch, freeing the greater curvature from attachments to the spleen and the splenic colon (ligating the gastric brevia). The stomach was then easily drawn through the diaphragm into the chest. The distal portion of the esophagus was resected, including the diverticulum and an anastomosis was effected between the stomach and the esophagus, terminolateral in type. The original cardiac opening of the stomach was closed, using interrupted black silk. The anastomosis was done in two layers with interrupted black silk. The wound was closed in layers leaving 50 c.c. of penicillin solution containing 100,000 units. One rubber tube drain through a stab wound in the eighth intercostal space posteriorly was used.

The patient received 500 c.c. of blood during the operative procedure, and he withstood the procedure well. The thoracotomy tube was connected to underwater drainage with a small amount of negative pressure. There was a minimal amount of drainage, the tube being removed on the fourth postoperative day. The temperature never went over 100.6° F., and the patient was started on full liquids on the fourth day and was on soft food on the fifth day, with no difficulty in swallowing. The patient was discharged entirely well on the twelfth postoperative day. Follow-up examinations have found the patient with no complaints, eating all food with no difficulty.

*Pathology:* Gross: Resected portion of esophagus with diverticulum. Microscopic examination showed leukoplakia with chronic ulceration, chronic inflammation, and some mucosal hyperplasia.

*Diagnosis:* Diverticulum and ulcer of lower portion of esophagus.

*Comment:* The operative procedure of resecting the diverticulum or diverticula and the distal portions of the esophagus has never been reported. It is definitely indicated in those patients who would require a diverticulectomy and also require a plastic repair of the esophagus for stricture or cardiospasm. The mobilization of the stomach into the chest and subsequent anastomosis is relatively simple now, so that there should be no hesitation in resecting the pathology completely.

### SUMMARY

All cases of epiphrenic diverticula of the esophagus recorded in the literature are reviewed. There were 126 cases reported in the literature. Of these, forty-three, or 33 per cent, were operated upon, with a mortality of 0.9 per cent. The transition of operative technique is discussed, and a new procedure, that of resecting the distal portion of the esophagus and cardia of stomach with intrathoracic esophagogastrostomy, is presented. Two case reports are given which utilized this operative procedure.

It is felt by the authors that this new operative procedure should be reserved for those cases that would not be adequately treated by diverticulectomy; namely, multiple diverticula, diverticula associated with cardiospasm, with esophageal atresia, or with neoplasm.

### ADDENDUM

Since this article was submitted, another successful case of diverticulum of the esophagus associated with carcinoma was successfully operated upon.

CASE 3.—Mr. J. C. (Flower-Fifth Avenue Hospital, No. 6044) a white man, aged 63 years, was admitted on June 13, 1951, because of difficulty in swallowing. The patient was first seen on June 11 and gave the following history:

*History:* For the past three months he had increasing difficulty in swallowing. Passage of soft food was very slow and though he never vomited, he has often felt like it. During the past three weeks, he complained of increasing difficulty in swallowing liquids with occasional regurgitation. Occasionally, he complained of substernal pain which was relieved by regurgitation. He lost at least 10 lbs. in the last three months.

*Physical Examination:* Patient is an elderly white male presenting a picture of dehydration and loss of weight. Blood pressure 110/80. Except for some external hemorrhoids, general physical examination was negative.

*Laboratory Data:* Urine examination was negative, except for a trace of albumin. Blood examination showed an average normal red cell count, with a hemoglobin of 75 per cent and W.B.C. 13,000, with normal differential. Blood Wassermann was negative. The blood chemistry was within normal range.

*Roentgenographic Examination* revealed an esophagus which was moderately dilated, with slight redundancy of the middle third. There was definite delay in passage of the barium through the lower end of the esophagus into the stomach. There was a marked irregular narrowing deformity of the lower end of the esophagus, with a moderate sized diverticulum-like pouch extending to the left of the midline. The possibility of a neoplasm could be ruled out (Fig. 5).

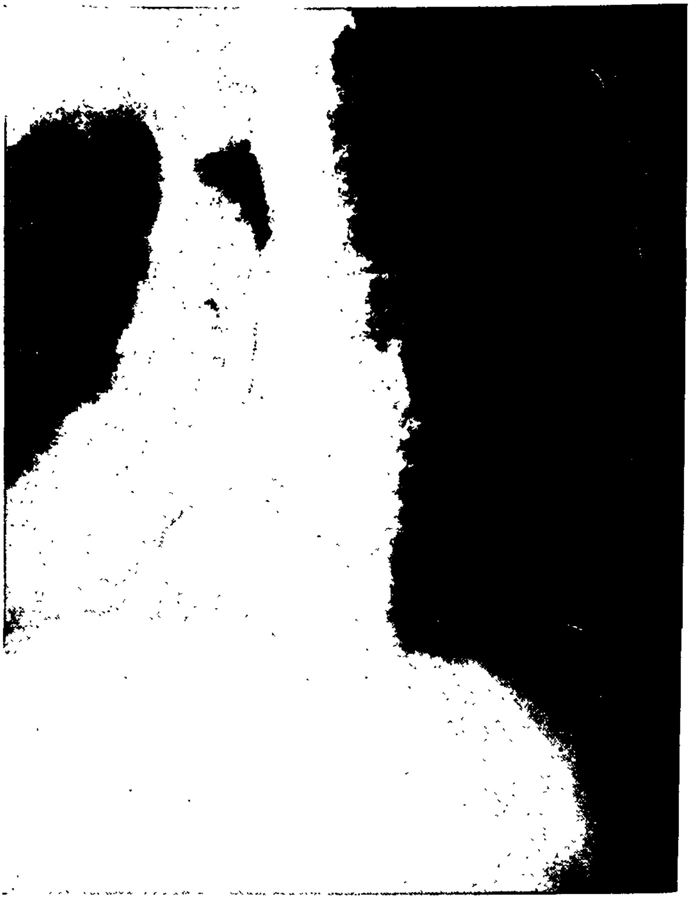


Fig. 5.—Case 3. Preoperative roentgenogram.

*Operation:* On June 15, 1951, an esophagogastrostomy was performed following the routine described above, resecting the lower half of the esophagus, together with the upper one-fourth of the stomach, including nearly the upper half of the lesser curvature. In performing the anastomosis, the anterior wall of the stomach was folded in below the site of the anastomosis to create a flap. The purpose of this flap was to help check gastric reflux into the esophagus. The anastomosis was about 2 c.c. in diameter when completed.

*Course:* Considering the magnitude of the operation, the patient had little reaction. He was allowed out of bed on the third postoperative day. There was no dyspnea at any time. The patient was allowed to swallow fluids on the third postoperative day. By the seventh postoperative day, he was on a regular diet. The patient was discharged on the tenth postoperative day. The patient was subsequently seen on September 25. He had no difficulty in swallowing, was working, and had gained 10 pounds. A postoperative roentgen examination revealed an esophagus normal in width at this time without obstruction to the flow of barium into the stomach. About 25 per cent of the stomach was above the left leaf of the diaphragm (Fig. 6).

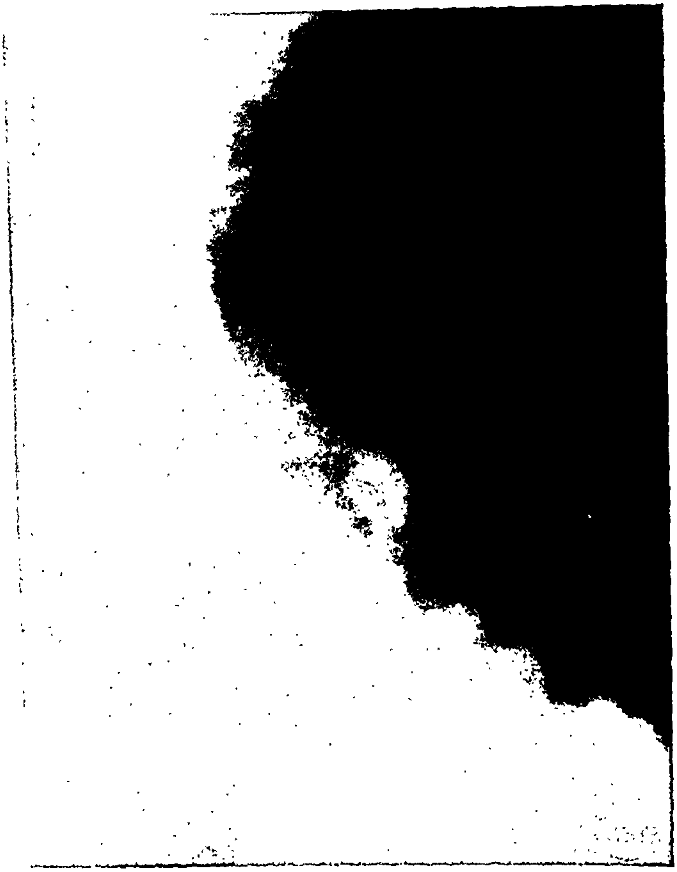


Fig. 6.—Case 3. Postoperative roentgenogram (close-up).

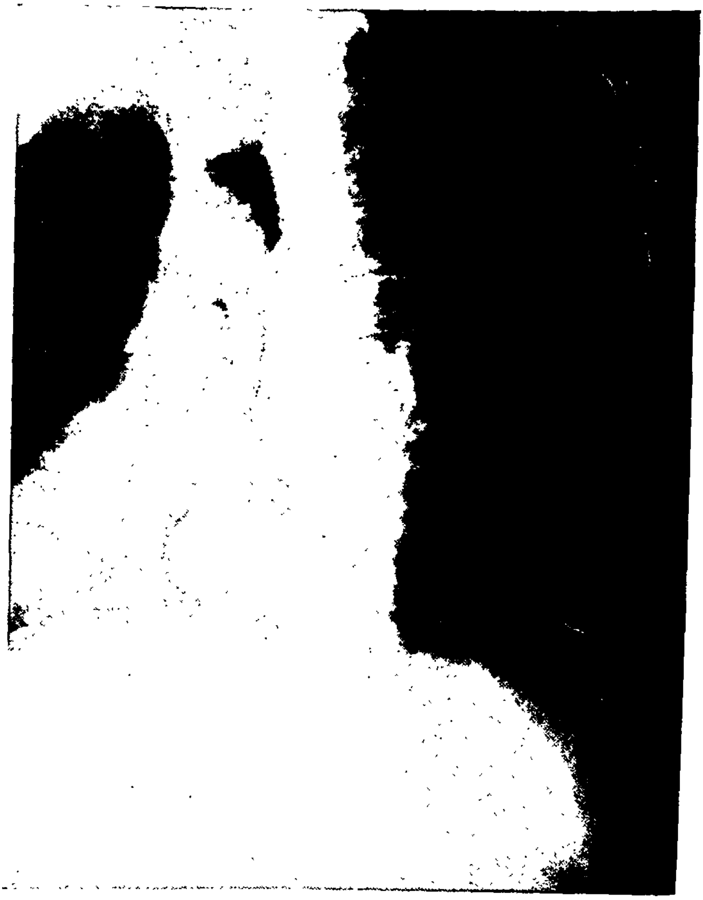
*Pathologic Report:* Specimen consisted of a long mass of esophageal tissue including the cardia and lesser curvature of the stomach. In the mid-portion a 1.5 cm. diverticulum leading into the esophagus was seen. A hard white firm mass, 7 by 4 cm. was seen encircling two-thirds of the esophagus not invading the lumen. Study of the proximal end of the resected esophagus showed almost 1.5 cm. of smooth muscle entirely free of invading carcinoma. At the level of the tumor, however, invading squamous cells had penetrated into the adventitial tissue of the esophagus. Study of the distal or cardiac end of resected specimen showed gastric mucosa and wall free of carcinomatous invasion. In one section the junction of gastric mucosa and squamous epithelium of the esophagus was seen. At this point there was slight invasion of the gastric musculature by the squamous carcinoma.

*Gross and Microscopic Diagnosis.—*

*Esophagus:* Epidermoid carcinoma showing good differentiation and invasion of all coats of the wall.

*Stomach:* No pathologic diagnosis.

*Roentgenographic Examination* revealed an esophagus which was moderately dilated, with slight redundancy of the middle third. There was definite delay in passage of the barium through the lower end of the esophagus into the stomach. There was a marked irregular narrowing deformity of the lower end of the esophagus, with a moderate sized diverticulum-like pouch extending to the left of the midline. The possibility of a neoplasm could be ruled out (Fig. 5).



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*Comment:* The diverticulum in this case is probably of the pulsion type. As a result of almost complete encirclement of the esophagus, a weakness in the wall had developed resulting in the formation of the diverticulum in response to the increase of intra-esophageal pressure.

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# SINGLE LEFT SUPERIOR VENA CAVA, WITH AORTIC COARCTATION, IN AN ADULT

## Case Report

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The occurrence of bilateral superior vena cava is a well-recognized anomaly. Sanders,[1] in 1946, found 215 such cases recorded in the medical literature. The existence of a left superior caval trunk, without a corresponding vessel on the right, is much less common. Atwell and Zoltowski,[2] in 1938, listed nineteen previously reported cases, and added one of their own. No report of a similar instance could be located in the English writings since that time.

The case reported here is that of a man who was found at autopsy to have a coarctation of the aorta, in close anatomic relationship to a single left superior vena cava.

## CASE REPORT

The patient, a well-developed muscular man of 47 years, who was stated to have been previously in good health, died suddenly and unexpectedly at home. In a complete autopsy, performed twelve hours after death, the significant findings were confined to the cardiovascular system and the lungs.

The heart was hypertrophied weighing 600 grams. This enlargement mainly involved the left ventricle, the myocardium of which was firm and measured 2.8 cm. in thickness. While the mitral, tricuspid, and pulmonary valves were thin and pliable, the aortic cusps were somewhat contracted, sclerotic, and nodular. The coronary arteries showed considerable narrowing by scattered, raised, atheromatous plaques, but there were no areas of complete occlusion. Just beyond the origin of the left subclavian artery there was a coarctation of the aorta. The lumen at the constricted point was 0.5 cm. in diameter. The intimal surfaces in this situation, and the proximal 2 cm. of the left subclavian artery were raised and roughened by yellowish plaques, many containing calcium. In contrast, the intima distal to the coarctation was smooth and the aortic wall elastic. Enlarged internal mammary and anterior intercostal arteries indicated a well-developed collateral circulation. Also of increased size were the first posterior intercostal arteries, arising from the aorta immediately distal to the coarctation.

Formed by the union of the left innominate and the longer, obliquely placed, right innominate vein, was a large left superior vena cava overlying the aortic arch and closely approximated to the groove produced by the coarctation (Fig. 1). This anomalous vein was continuous with a vascular channel contained within the posterior wall of the left atrium, draining into the right atrium. No superior caval trunk was present on the right side.

In addition to these abnormalities of the heart and great vessels, the lungs were heavy, wet and subcrepitant, the left weighing 720 grams and the right 860 grams. The other organs were essentially unremarkable grossly.

Microscopic examination revealed little worthy of mention, aside from congestion and edema of lungs.

## COMMENT

Consideration of the normal development of veins from the cardinal system indicates the series of events leading to the establishment of a single superior caval trunk on the left side. According to Arey,[3] during the sixth week of

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intrauterine life, the true precardinal veins, located laterally in the segmented portion of the head, drain into the common cardinal veins of each side. Normally, an oblique anastomosis occurs at about the eighth week, draining from the left to the right precardinal. This anastomotic trunk becomes the left innominate vein of the adult and the left precardinal persists merely as part of the highest intercostal vein on that side. The stump of the left common cardinal normally assists in forming the coronary sinus and sometimes the oblique vein of Marshall. It is seen that the superior vena cava is usually formed on the right side by the common cardinal and precardinal veins as far as the intercardinal anastomosis (Fig. 2*A*). In this case, a reversal of the normal development evidently occurred, the superior vena cava being formed by the left common cardinal and precardinal veins and draining into the right atrium through a vascular channel contained within the wall of the left atrium (Fig. 2*B*).

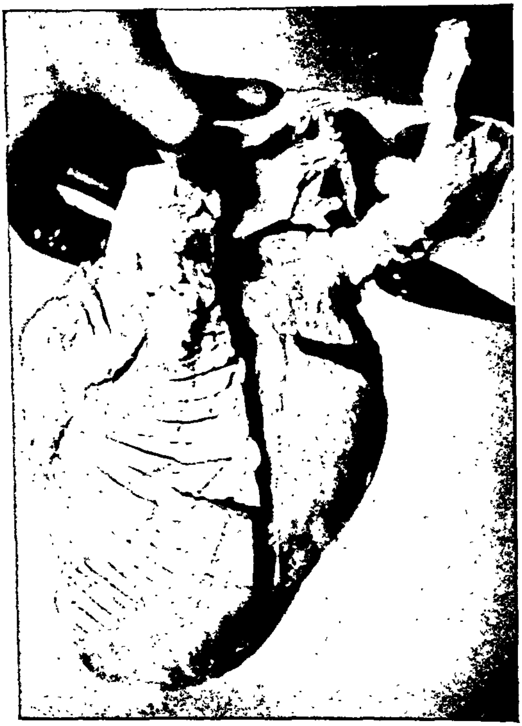

Fig. 1.—Heart, aortic arch, and left superior vena cava. The latter has been displaced laterally to show the aortic coarctation, over which it lay.

A feature of major interest in this case was the presence of both a left superior vena cava and coarctation of the aorta. The association between aortic coarctation and anomalies of the heart and great vessels is well known. The relationship is so frequent as to suggest to Hamilton and Abbott[4] a significant anatomical complex. In an exhaustive statistical report on 200 cases of coarctation of the aorta in 1928, Abbott[5] found 111 cases in which there were other cardiac or vascular malformations. However, the persistence of a left superior vena cava was reported in only three cases. In these it is not clear whether the right vena cava was also present. Reifenstein, Levine, and Gross[6] recently reviewed the literature on aortic coarctation since Abbott's publication, collecting 104 cases. They listed fourteen types of cardiovascular anomaly occurring in association with coarctation of the aorta but did not encounter a case of persistent left superior vena cava.

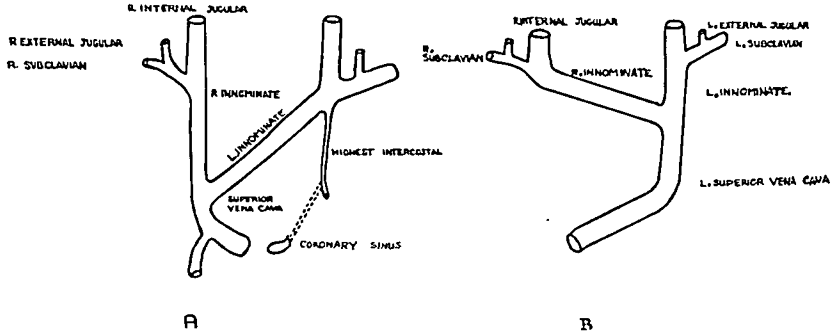

Fig. 2.—*A*, Diagram to show the normal adult arrangement of veins derived from the cardinals (adapted from Arey, L. B.: *Developmental Anatomy*). *B*, Diagram of the arrangement of veins in the present case. There has been a reversal of the normal development, with a resultant left superior vena cava.

Concerning the etiology of coarctation of the aorta, a relationship with the ductus arteriosus, as suggested by the anatomic site of the narrowing, has been widely accepted. According to Hamilton and Abbott, a theory based on this relationship was first proposed by Craigie in 1841. To him it seemed likely that the obliterating potentialities of the ductus arteriosus had for some peculiar reason been extended into the aorta and there given rise to contraction of the wall of that vessel. Hamilton and Abbott believed this explanation (called the Skodaic theory after Skoda who supported it) to be true, at least in part, although the hypothesis of Brunner that in these cases the peculiar tissue of the ductus wall is continued into the wall of the aorta has never been substantiated. It is also of interest that aortic coarctations occur most commonly at a site immediately distal to the origin of the left subclavian artery. It is at this point that the embryonic right arch is detached during the development of the main left dorsal aorta. Here, again, some obliterative process may be proposed as the cause of narrowing.

Despite theories which attempt to relate the development of coarctation to obliterative vascular processes, importance has also been attached to the mechanical effect of traction of the fibrous obliterated ductus arteriosus on the arch of the aorta. Hamilton and Abbott state that this has sometimes been so strong as to produce a tent-shaped aneurysm of the aortic wall in this region. In the present case, the close anatomical relationship of the large left superior vena cava to the coarctation causes speculation as to what effect mechanical pressure might have had on initiating or augmenting the constructive process in the aorta.

In view of the present-day surgical treatment of coarctation of the aorta, any anomaly of possible surgical significance seems worth stressing. Lam,[7] in 1945, recorded a case in which a large anomalous inferior vena cava interfered with an operation for patent ductus arteriosus. It appears likely that, in the case here presented, surgical treatment of the coarctation might well have been impossible. The fact that a persistent single left superior vena cava is rare in association with coarctation has been amply indicated above. Nevertheless, in an individual case, its presence must necessarily be of vital importance in surgical treatment.

It is necessary to give consideration to the cause of death in this case. It seems probable that death was in some way related to the aortic lesion. The persistent left caval trunk could not have had any lethal influence and a complete autopsy failed to reveal any lesion of importance outside the cardiovascular system. Reinfenstein and his co-workers, in 104 cases, found death to be due to rupture of the aorta in 23.1 per cent, to bacterial endocarditis or aortitis in 22.1 per cent, to congestive heart failure in 18.3 per cent, to intracranial causes in 10.6 per cent and to unrelated causes such as pneumonia and carcinoma in 25.9 per cent. In the last category they included three cases in which death was sudden and attributed to coronary arterial disease, and two cases in which death was sudden and unexplained. In the case reported here, the heart was markedly hypertrophied, weighing 600 grams. The coronary arteries showed areas of atheromatous thickening but there were no regions of marked stenosis or occlusion. It seems probable that death in this instance was due to relative myocardial ischemia.

## SUMMARY

A case, in a man of 47 years, of a single left-sided superior vena cava associated with coarctation of the aorta is reported. This apparently brings to twenty the number of recorded cases in which a left superior caval trunk occurred without a corresponding vessel on the right side. It would appear that the association of a left superior vena cava with aortic coarctation has been observed on but 3 previous occasions. In these three recorded instances, it is not known whether there was also a right superior vena cava. It is suggested that this type of anomaly may be of importance in the surgery of aortic coarctation.

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# CARCINOMA OF THE TRACHEA

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RECENT reports of the laboratory investigation of various methods of bridging gaps in the trachea and bronchi are indicative both of an interest in the treatment of tracheal tumors and the lack of a definitive method of reconstruction which can be applied to human patients with primary tumors of the intrathoracic trachea. Belsey has published a review of the problem which includes a report of two cases in which he partially resected a considerable segment of the intrathoracic trachea and carried out a reconstruction with fascia lata over a framework of stainless steel wire, with brilliant initial success. The only report of a successful initial result after resection of a complete segment of the intrathoracic trachea in a human subject is that of Jarvis, who bridged the gap with a stainless steel tube in the manner evolved by Daniels in experimental work on dogs. Clagett attempted a similar reconstruction, using a tube of polyethylene, but his patient died.

Because primary malignant tumors of the trachea are rare, no one surgeon is likely to have an opportunity to treat more than a very few. For that reason the report of a single case may be justified. The method of reconstruction used in this case failed, but the principle involved appears to be sound; the failure was due to technical details which are capable of correction.

G. E., a white man aged 51 years, was admitted to the medical wards of the Toronto General Hospital on June 30, 1949, with a diagnosis of acute pulmonary edema. For three months he had had a severe cough, productive of mucoid sputum, with marked shortness of breath and orthopnea. On the day before admission he had coughed up a large amount of blood. He had lost forty pounds. He was a chronic alcoholic who for years had had cough with moderate sputum.

The patient was an obese man with a short neck and deep chest. Even in an oxygen tent he showed extreme dyspnea with persisting cyanosis, but no evidence of cardiac failure. Coarse rhonchi were present throughout the chest. He developed clinical and radiographic evidence of bilateral bronchopneumonia with a fever up to 103° F., which did not respond to penicillin, and continued to cough up large amounts of blood-tinged sputum.

Laminagrams (Fig. 1) demonstrated a rounded mass arising from the right side of the trachea just above the right main bronchus and almost completely occluding the lumen of the trachea. A bronchoscopic examination on

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July 25 revealed a mass bulging from the right wall of the lower trachea, and below this the right main bronchus could be seen containing granulation tissue. A biopsy was taken but failed to show tumor. On withdrawing the bronchoscope the trachea became completely obstructed so a gum elastic catheter was passed into the right main bronchus. This was left in place for five days and then removed, and the patient continued to have a fairly adequate airway.

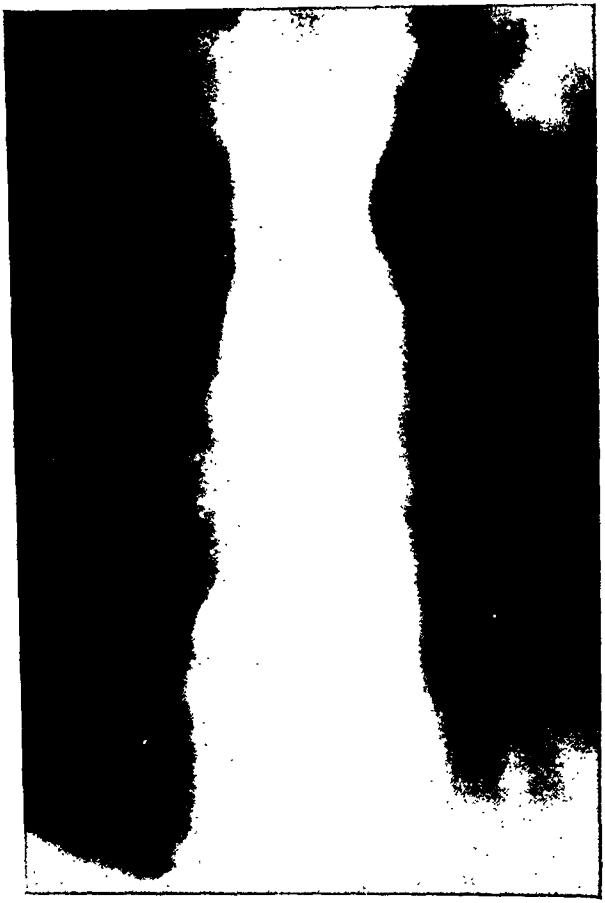

Fig. 1.—Laminagram showing tumor arising from right wall of lower trachea and almost completely obstructing lumen.

At this point the patient was seen by the surgical service. He had bilateral bronchopneumonia, with a temperature of 102° F. There was a persistent substernal wheeze, cough with copious sputum, and he seemed in imminent danger of recurrence of tracheal obstruction and death from asphyxia. It was decided to attempt removal of the tracheal tumor.

On August 5 the patient was anesthetized with pentothal and curare and, using a bronchoscope, an endobronchial catheter was forced past the growth into the left main bronchus, and the anesthetic continued with cyclopropane.

The right chest was opened by resection of the sixth rib. The posterior half of the lower lobe including the superior segment showed consolidation. The azygos vein was divided, and the trachea exposed. On the right side, just above the main bronchus, a tumor could be felt, about 2.5 cm. in length. Adherent to the trachea in this area was a lymph node, 1 cm. in diameter, which was removed and showed no tumor on frozen section. At this point the patient was doing badly. He was very anoxic, the heart had stopped, and had restarted after massage. The tumor was rapidly excised, leaving a defect in the trachea 3 cm. by 1 cm. This was covered by a free graft of pericardium held taut by interrupted sutures of fine silk, and covered by a strip of Gelfoam. The chest was closed with underwater drainage.

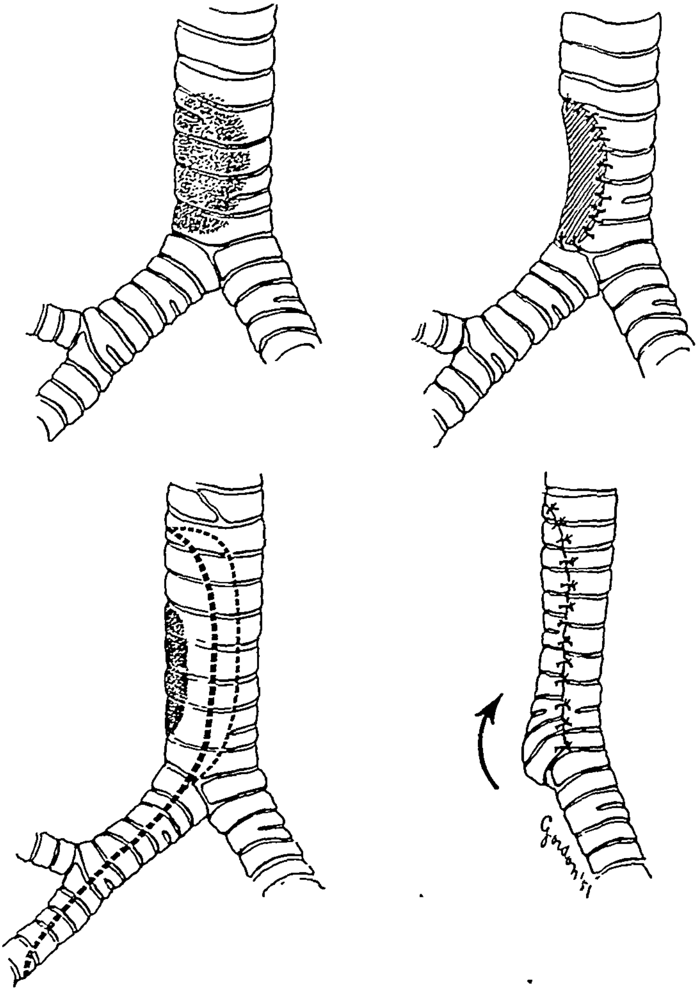

Fig. 2.—The upper diagrams illustrate the position of the original lesion, and the repair of the defect with a patch of pericardium after local excision. The lower diagrams illustrate the treatment of the recurrence and the reconstruction of the trachea using a pedicle graft from the right bronchus.

The patient made a remarkably good recovery. The bronchopneumonia cleared over the course of five days, and following that the patient had only his usual chronic cough, and a good airway.

Examination of the tissue removed from the trachea revealed a typical squamous-cell carcinoma with many epithelial pearls.

It was obvious that this local procedure could not cure this lesion. Commencing on September 10 a course of roentgen therapy was given, centered on the lower end of the trachea from multiple ports, with a tumor dosage of 3,100 r. units. While this therapy was still being given a further bronchoscopic examination, two months after operation, showed a small area of recurrence, which was confirmed by biopsy. It was decided that a further surgical excision must be attempted.

On November 5 the patient was again anesthetized but immediately went into a state of collapse with systolic blood pressure of 60 mm. A diagnosis of coronary infarction was made, and the operation postponed. This diagnosis was confirmed by electrocardiographic examination. The patient made a good recovery.

On November 25 anesthesia was again induced and a large endobronchial catheter passed into the left main bronchus. The right chest was reopened, the lung freed and the trachea examined. There was considerable inflammatory reaction at the site of the previous operation, but no evidence of tumor outside the trachea. It was decided that because of the proximity of the tumor to the right main bronchus an adequate resection would involve sacrifice of the right lung. The pulmonary artery and veins were divided, and all the hilar structures freed except the bronchus. An incision was then made in the trachea and carried down on to the right main bronchus in such a fashion as to remove two-thirds of the circumference of the trachea for a distance of 5 cm. above the bifurcation, together with the lung and the upper half of the main bronchus, but preserving the lower half of the right main bronchus for a similar distance of 5 cm. In this way a pedicle graft of bronchus was created to correspond to the defect in the trachea, hinged at the carina. It was easily turned upward over the defect in the trachea, fitted it accurately, and was sutured into place with multiple interrupted sutures of fine silk. A wide resection of mediastinal pleura and areolar tissue about the trachea had been done, so no tissue was available to cover the graft. The chest was closed with underwater drainage.

The operation was tolerated well and the patient did extremely well for two days. Air entry into the left lung was good, there was no evidence of tracheal obstruction, and he was able to raise considerable amounts of tenacious mucus. On the third day he suddenly coughed, was unable to clear the mucus and died immediately of asphyxiation. Just as he died a bronchoscope was passed, and it was found that the lower trachea was oval in shape but a 9 mm. bronchoscope was easily passed into the left main bronchus which was completely occluded with thick mucus.

Post-mortem examination confirmed the obstruction of the left main bronchus by a mucous plug. The anterior descending branch of the left coronary artery was obstructed by organized thrombus, and there was a large healing infarct of the anterior wall of the left ventricle. No residual carcinoma could be demonstrated. The bronchial graft was alive and apparently healing.

## DISCUSSION

A large proportion of primary tumors of the trachea occur at or near the lower end so that reconstruction using the right main bronchus may be considered as one of the possibilities. Belsey laid down four characteristics which are essential to a satisfactory method of reconstruction as follows: (1) lateral rigidity; (2) longitudinal elasticity and flexibility; (3) an adequate and air-tight lumen; (4) an uninterrupted lining of ciliated columnar epithelium. The method which has been described fulfilled the last three requirements very well but was somewhat deficient in regard to the first. In this case the right half of the cartilaginous portion of the trachea was removed together with the whole membranous portion. The pedicle graft was in itself of normal rigidity but could hinge at the two suture lines, anteriorly and posteriorly, and so assume a scabbard shape. The lumen was adequate for respiration, and accepted a 9 mm. bronchoscope, but probably tended to collapse with cough, and this undoubtedly contributed to the difficulty in raising mucus. Splinting the lumen in a circular shape by the use of wire, or the temporary use of an intracheal tube until healing was sound would not be a difficult addition to the technique.

Both Jarvis and Clagett performed tracheotomy on their patients to permit easy and certain removal of secretions by suction. That was not done in this case, and tracheal suction by intubation through the nose proved difficult and unsatisfactory. It is probable that the patient would have survived if it had been possible to remove secretions efficiently. Since any method of reconstruction is likely to result in both increased secretion and some disturbance of the cough mechanism, a tracheotomy is a simple and wise precaution.

## SUMMARY

A case of squamous-cell carcinoma of the lower end of the trachea is reported. As an emergency measure, to overcome tracheal obstruction, the tumor was excised locally and the defect successfully repaired with a patch of pericardium. A later radical resection with reconstruction using a pedicle graft of bronchus ended in failure. The technical errors responsible for failure are discussed.

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# BRONCHIAL OBSTRUCTION WITH CHRONIC ATELECTASIS AND PNEUMONITIS DUE TO HILAR LYMPHADENITIS AFTER LOBECTOMY AND LINGULECTOMY

## A Case Report

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THE syndrome of chronic atelectasis and pneumonitis most often involving the middle lobe is well known, and has been adequately described in the papers of Graham, Burford, and Mayer,[4] and Paulson and Shaw.[6] That other lobes and segments are sometimes involved is shown by the reports of several surgeons.[1, 3, 5, 6] Clinically, there is usually a rather prolonged history of chronic pulmonary infection with cough, pain, dyspnea, hemoptysis, and wheezing. Atelectasis of the involved pulmonary tissue and sometimes calcified hilar lymph nodes are seen on roentgenograms. Narrowing of the bronchus may be demonstrated by lipiodol studies and bronchoscopy.

At operation most of these patients are found to have bronchial obstruction of varying degree usually secondary to compression of the bronchus by enlarged hilar lymph nodes. This lymph-node enlargement may be caused by infection elsewhere than in the portion of the lung distal to the obstruction. In a few cases, the bronchial obstruction is due to other factors such as foreign body or intrinsic bronchial disease. Brock and associates[2] have described the anatomic peculiarities of the middle-lobe bronchus which apparently account for the relatively frequent involvement of the middle lobe.

The case to be reported here is one in which atelectasis and chronic pneumonitis, developing in the remaining part of the left upper lobe a few weeks after left lower lobe lobectomy and lingulectomy, necessitated removal of the remainder of the left lung.

### CASE REPORT

This 8-year-old white boy was admitted to Jefferson Hospital on the service of Dr. Louis H. Clerf. The history was that of chronic suppurative pulmonary disease of five years' duration. Three months prior to admission he had had a severe attack of laryngo-tracheobronchitis after which he had remained hoarse and dyspneic. Upon physical examination, he was observed to be thin, undernourished, and hoarse. An obstructive type of dyspnea markedly limited physical activity. There was slight cyanosis of the fingernails, but no clubbing. His cough was productive of large quantities of purulent sputum. With the direct laryngoscope, there was seen marked edema about the left arytenoid cartilage and redundancy of the overlying mucosa. The bronchoscopist observed much purulent secretion in both bronchi. Increased difficulty in breathing followed the bronchoscopy, and a tracheotomy was performed. A plain roentgenogram of the chest was not abnormal. A bronchogram demon-

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strated unilateral saccular bronchiectasis involving all segments of the left lower lobe and both segments of the lingula of the left upper lobe. The remainder of the tracheobronchial tree was well outlined, and appeared normal.

He was transferred to the surgical service, and a left lower lobe lobectomy and lingulectomy was performed without incident. The remainder of the left upper lobe seemed normal except for a few overlying adhesions. There was no unusual enlargement of the hilar lymph nodes. Of possible importance in the light of later events is the fact that after closure the bronchial stumps were reinforced by suturing over them mediastinal tissues and lymph nodes.

His immediate postoperative course was entirely satisfactory. Roentgenograms of the chest on the first, third, and ninth postoperative days showed satisfactory expansion of the remainder of the left upper lobe. The lung parenchyma appeared normal (Fig. 1). He became afebrile quickly, and his sputum was greatly diminished and no longer purulent. He was returned to Dr. Clerf's service for further treatment of his laryngeal lesion and removal of the tracheotomy tube.

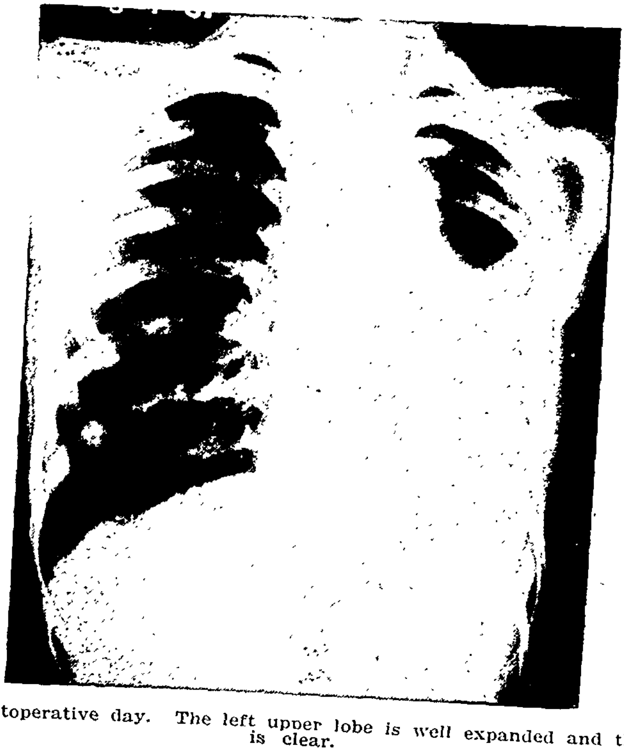

Fig. 1.—Ninth postoperative day. The left upper lobe is well expanded and the parenchyma is clear.

Three weeks after operation, however, his cough again became troublesome and productive of purulent secretions. He developed a septic type of fever. At bronchoscopy a week later, pus was seen coming from the left upper lobe bronchus which appeared distorted. Repeated bronchoscopic examinations during the ensuing four weeks revealed increasing stenosis of the left upper lobe bronchus progressing to the point of almost complete obstruction. At the same time, the purulent secretions increased and his general condition deteriorated. A roentgenogram showed the left hemithorax to be obscured, and the left upper lobe to be only partially aerated (Fig. 2). Instilled lipiodol puddled in the left upper lobe and the narrowing of the bronchus was visualized (Fig. 3).

A second thoracotomy was performed approximately nine weeks after the original operation. There was no free fluid, and the pleural space was obliterated by edematous adhesions. The remainder of the left upper lobe appeared chronically infected and almost completely atelectatic. Of most interest was a fixed group of enlarged lymph nodes adherent to one another and so situated as to form a constricting ring about the left upper lobe bronchus markedly compressing it. A left upper lobe lobectomy was done and most of the left main bronchus removed. The patient's convalescence from this operation was uneventful, and he is again afebrile and almost free of secretions. Eight weeks after the operation, his general condition is decidedly improved and early removal of the tracheotomy tube is anticipated.

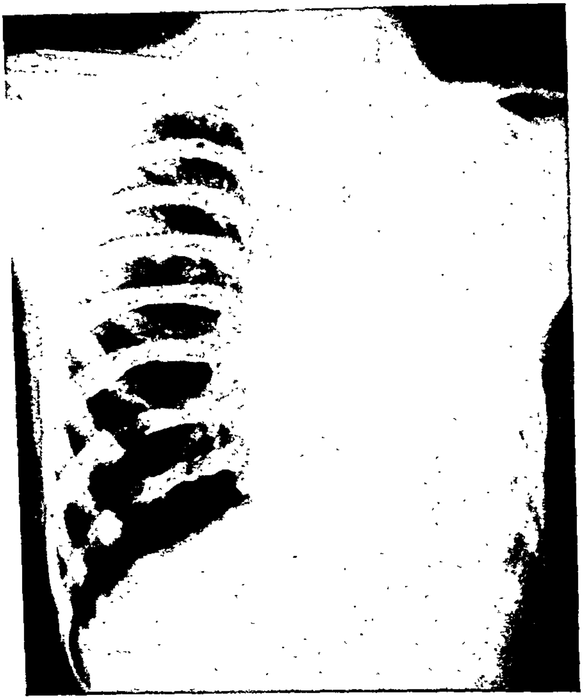

Fig. 2.—Fifty-fifth postoperative day. The left hemithorax is obscured and the left upper lobe partially atelectatic.

Dissection of the operative specimen confirmed the impression gained at operation that the bronchial obstruction was due entirely to compression by the encircling lymph nodes. The two bronchial stumps remaining from the original lobectomy and lingulectomy were well healed, and in on way encroached upon the lumen of the bronchus. There was no intrinsic obstructive lesion of the bronchus at the point of constriction and, after separation of the encircling nodes, the lumen appeared normal. The lobe itself was atelectatic and chronically infected with cylindrical bronchiectatic changes in the bronchi. No abscess cavity was present. The encircling lymph nodes and parenchymal changes are demonstrated in Fig. 4.

## DISCUSSION

The case presented here is one in which obstruction of the bronchus to the remaining portion of the left upper lobe after left lower lobe lobectomy and lingulectomy was apparently due to external compression from a ring of enlarged lymph nodes. That the atelectasis and chronic pneumonitis probably followed this obstruction is indicated by the very satisfactory course in the early postoperative period and the absence of any indication of lesions in the

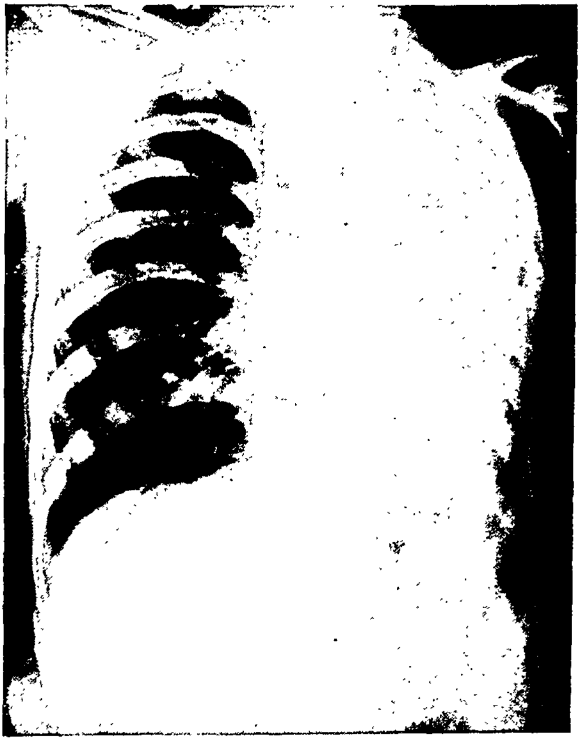

Fig. 3.—Fifty-seventh postoperative day. The lipiodol has pooled in the left upper lobe and the bronchus is narrowed.

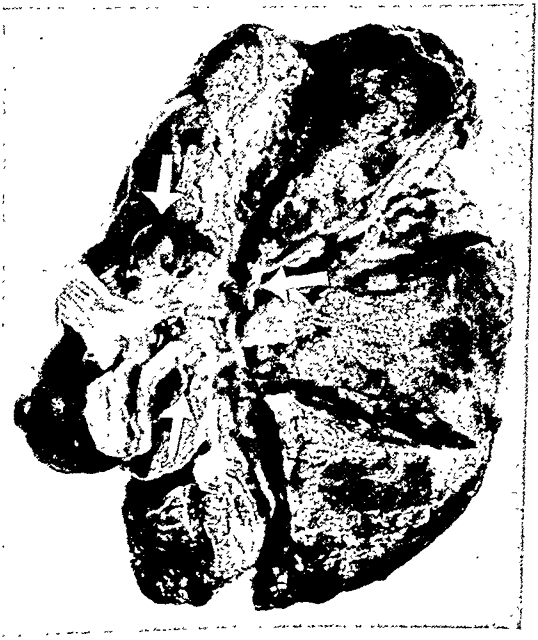

Fig. 4.—The operative specimen. Some of the enlarged lymph nodes encircling the left upper lobe bronchus are indicated by arrows. Others lie behind the bronchus.

left upper lobe outside the lingula on the preoperative roentgenograms, at the first operation, or on the roentgenograms during the early postoperative period. The early distortion of the bronchus was not due to displacement of the upper lobe, since peripheral adhesions undivided at the first operation effectively held it in the proper position. The closure of the left lower lobe and lingular bronchi in no way encroached upon the bronchus, nor was there any obstructing lesion of the bronchus itself.

In retrospect, it is probable that the method of covering the bronchial stumps with lymph nodes may have contributed to the formation and fixation of the lymphatic ring. Subsequent enlargement of the nodes secondary to the inflammatory response to the operation could then have caused pressure and obstruction. It is possible that earlier thoracotomy might have permitted relief of the obstruction before serious damage to the pulmonary tissue made excision mandatory.

The possibility of this unusual complication should be borne in mind during the performance of pulmonary lobectomy when tissues to reinforce the bronchial stump are being selected so that the formation of a fixed lymphatic ring about the bronchus may be avoided.

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# RETINAL ISCHEMIA WITH UNILATERAL BLINDNESS—A COMPLICATION OCCURRING DURING PULMONARY RESECTION IN THE PRONE POSITION

## Report of Two Cases

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Oteen, N. C.

SINCE the introduction of the prone position for thoracic surgical procedures in 1946, its use has become widespread both in this country and abroad. Many types of apparatus are available for converting the standard operating table for adaptation to this operating position. A special Overholt-Comper Thoracic Table has been made available by the American Sterilizer Company which, in our experience, has been the most satisfactory. In all of these modifications the principle is the same in that head and pectoral supports brace the corresponding anatomic parts. A pelvic support and the body of the table bear the weight of the pelvic girdle and lower extremities. In all instances the head support is horseshoe shaped and is cushioned with rubber. This throws the weight of the head on the frontal bone and the zygomatic processes.

It is the purpose of this paper to present two cases of unilateral blindness following pulmonary resection in the prone position. In each case blindness was attributed to pressure applied to the globe with resultant increased intraocular tension and ultimate ischemia of the retina. This catastrophe was caused by an undetected rotation of the head during the course of the long operative procedure. Direct pressure from the head rest was placed on the globe for a protracted period of time, and loss of vision through retinal destruction resulted.

Case 1.—F. F., a 28-year-old Negro man, who underwent a right pneumonectomy for pulmonary tuberculosis on Nov. 17, 1949. The operation started at 8:30 A.M., and terminated at 11:30 A.M. The operative procedure per se was uncomplicated. On removal of the patient from the Overholt-Comper Table, it was noted that a marked exophthalmus was present in the right eye. An ophthalmologist was immediately consulted and the following report was made:

> Ischemia of macular area. Arteries attenuated, but not typical of central artery occlusion. Marked exophthalmus present. Permanent destruction of the retina has occurred.

It was the opinion of this consultant that emergency measures could not alter the status of vision in the right eye. By the tenth postoperative day the exophthalmus had subsided. A careful follow-up during the next few months showed a slight return of light perception, but total blindness persisted to all other stimuli.

Case 2.—C. L., a 27-year-old Negro man, who underwent a right upper lobectomy for pulmonary tuberculosis on May 18, 1951. The operation started at 8:30 A.M., and terminated at 11:50 A.M. No complication occurred during the course of the operative procedure. On removing the patient from the Overholt-Comper Table, it was noted that a marked exophthalmus was present in the right eye, with unusual injection of the scleral vessels. Opthalmologic examination was obtained, and the following report was made:

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the results of their own study and do not reflect the opinion of the Veterans Administration.

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Patient has complete occlusion of vessels in the right eye. All vessels appear to be empty. The optic disk is very pale. Tension is normal to palpation. There is no hope for return of vision in this eye.

The exophthalmus persisted for approximately one week, gradually subsiding over this period of time. No return of vision occurred on follow-up examination.

In reviewing the literature, no cases of traumatic blindness could be found with an etiologic basis resembling the preceding cases. However, the experimental studies by Kukan, quoted by Puntenney,[1] were encountered during this review, and his findings may throw some light on the mechanism involved in the production of blindness in these cases.

Kukan devised a method of ophthalmodynamometry that consisted of a negative pressure system involving a manometer, sucking disk, and suction pump. With this apparatus positive pressure could be applied to the eyeball when the sucking cup was held in apposition to the globe and negative pressure applied. He demonstrated mathematically that the pressure which sucks the cup simultaneously delivers pressure to the rim of the cup; when the surface area of this sucking portion exceeds that of the rim compression occurs and the intraocular tension rises. After compression has been maintained for a short period of time the aqueous fluid is forced out of the anterior chamber and the retinal blood flow is markedly diminished, with a subsequent drop in the intraocular pressure. It seems plausible that a similar mechanism of action could occur with prolonged constant pressure applied to the globe from the weight of the head compressing the eyeball against the cushioned head rest. A diminished retinal circulation with subsequent destruction of the retinal structures would be a possible sequence of events.

The preceding cases were presented to call attention to a serious complication that can occur with the use of the prone position in thoracic surgery. It is the feeling of the authors that the prevention of this complication will depend on close cooperation between the surgeon and anesthesiologist. It should be the combined duty of the surgeon and anesthesiologist to see that the head is placed properly in the head rest at the beginning of the operative procedure. The anesthesiologist should see that it remains in the proper position until the conclusion of the operation.

## SUMMARY

1. Two cases of unilateral blindness are presented occurring during pulmonary excisional surgery with employment of the prone position.
2. A possible mechanism of action is offered based on opthhalmodynamometric studies by Kukan.
3. Cooperation between the surgeon and anesthesiologist is urged in the prevention of this serious and avoidable complication.

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# COMBINED STREPTOMYCIN AND TUBERCULIN THERAPY IN EXPERIMENTAL TUBERCULOSIS

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THE investigation of Cairns and associates[1] suggest that the administration of tuberculin (PPD) and streptomycin together might prove beneficial. They treated patients suffering from tuberculous meningitis with tuberculin and streptomycin given intrathecally.

We wish to report the results of streptomycin and tuberculin (PPD) therapy in guinea pigs experimentally infected with tuberculosis. Suboptimal doses of streptomycin were employed. The dose of PPD employed in most instances limited the local tuberculin reaction to an erythematous reaction of slight to moderate intensity.

## METHODS

Fifty-six guinea pigs of mixed breed stock, weighing an average 316 Gm., were infected subcutaneously in the right groin region with 0.01 mg. (wet weight) of a 14-day-old transplant of human tubercle bacilli (strain H37rv), grown on egg medium. The inoculum was contained in 0.5 ml. of phosphate buffer solution of pH 7.8. The animals were divided into four groups. There were seven males and seven females in each group. There were a similar number of light colored animals in each group.

*Group A:* Fourteen guinea pigs were used as controls.

*Group B:* Fourteen guinea pigs were treated with PPD alone. Treatment was started two weeks following infection. The dose of PPD varied from 0.00001 mg. to 0.001 mg.

*Group C:* Daily, except Sundays, fourteen guinea pigs received 5 mg. of streptomycin subcutaneously for a period of six weeks. Treatment was started two weeks following infection. By this time the local lesion was well defined, the regional nodes palpable.

*Group D:* Fourteen guinea pigs were treated with streptomycin and PPD. Treatment was started two weeks following infection. The dose of streptomycin was 5 mg. daily; the dose of PPD varied from 0.00001 mg. to 0.001 mg.

Intracutaneous tuberculin injections were given at three- to four-day intervals. When the erythematous skin reaction occurred, it became evident within twenty-four hours. In most instances the reaction had subsided by seventy-two hours. When the more severe hemorrhagic necrotic type of local reaction developed, the acute phase seemed over within seventy-two hours. Repeated injections gave rise to fresh local responses, but did not seem to alter the appearance of the previous response.

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TABLE I. SUMMARY OF MACROSCOPIC TUBERCULOSIS FOUND IN EACH GROUP

| ANIMAL GROUP | GUINEA PIG | DEGREE OF MACROSCOPIC TUBERCULOSIS | | | | | |
|---|---|---|---|---|---|---|---|
| | | SPLEEN | LIVER | LUNGS | LYMPH NODES | | LOCAL LESION |
| | | | | | R.I. ⊙ | TB ⊙ | |
| Controls | (1) | +++ | +++ | ++-+++ | ± | ++ | +-++ |
| | (2) | +++ | +++ | ++-+++ | + | ++ | ++ |
| | 3 | +++ | +++ | ++ | + | ++ | +++ |
| | 4 | +-++ | ++-+++ | + | + | + | ++-+++ |
| | 5 | ++ | ++ | ++ | ± | ++ | ++ |
| | 6 | ++-+++ | ++-+++ | +-++ | ± | +-++ | +-++ |
| | 7 | ++-+++ | ++ | + | +-++ | +-++ | + |
| | 8 | +++ | +++ | + | + | + | + |
| | 9 | +-++ | - | - | ± | + | + |
| | 10 | ++-+++ | ++-+++ | +-++ | +-++ | +-++ | + |
| | 11 | +++ | ++-+++ | +-++ | ++ | ++ | + |
| | 12 | ± | + | +-++ | + | +-++ | ++ |
| | 13 | +++ | +-++ | ± | + | +-++ | + |
| | 14 | +-++ | ++-+++ | + | + | ++ | +++ |
| Tuberculin alone | (1) | ++ | ++-+++ | +-++ | + | + | ++ |
| | (2) | ++-+++ | +++ | ++-+++ | ± | ± | ++ |
| | (3) | +++ | +++ | +++ | + | + | ++ |
| | (4) | +++ | +++ | ± | + | +-++ | +++ |
| | (5) | +++ | ++-+++ | ± | + | + | ++ |
| | 6 | ++-+++ | +++ | ++-+++ | + | +-++ | +++ |
| | 7 | +++ | +++ | ± | + | +-++ | ++ |
| | 8 | +++ | ++ | ++-+++ | + | +-++ | ++ |
| | 9 | ++ | ++ | ± | ± | ± | + |
| | 10 | ++-+++ | ++ | + | + | +-++ | ++ |
| | 11 | +-++ | ++ | - | + | +-++ | ++ |
| | 12 | ++-+++ | ++ | ++ | + | + | +-++ |
| | 13 | ++ | ++ | +-++ | + | + | +-++ |
| | 14 | ± | + | ± | + | ++ | + |
| Streptomycin alone | 1 | - | ± | ± | + | ± | ± |
| | 2 | + | ± | - | ± | + | ± |
| | 3 | - | - | - | ± | ± | ± |
| | 4 | +-++ | - | + | + | ± | - |
| | 5 | - | - | - | ± | - | - |
| | 6 | ± | ++ | - | ± | ± | ± |
| | 7 | - | - | - | ± | ± | ± |
| | 8 | - | ± | - | + | - | - |
| | 9 | ± | - | + | ± | ± | - |
| | 10 | ± | - | - | ± | ± | ± |
| | 11 | ± | ± | ± | + | - | - |
| | 12 | + | + | - | + | ± | ± |
| | 13 | ± | - | - | ± | ± | ± |
| | 14 | ± | - | - | ± | - | ± |
| Streptomycin and tuberculin | 1 | - | - | - | ± | ± | ± |
| | 2 | - | - | - | + | - | - |
| | 3 | - | - | - | ± | - | - |
| | 4 | - | - | - | + | - | ± |
| | 5 | - | - | - | ± | ± | ± |
| | 6 | - | ± | - | + | ± | ± |
| | 7 | ± | - | ± | ± | ± | ± |
| | 8 | ± | - | - | + | ± | - |
| | 9 | - | - | - | + | - | - |
| | 10 | ± | ± | - | ± | - | ± |
| | 11 | - | ± | - | ± | ± | ± |
| | 12 | - | - | ± | ± | ± | - |
| | 13 | - | - | - | ± | - | - |
| | 14 | - | - | - | ± | - | - |

− = none or questionable tuberculosis.
± = slight.
+ = Moderate.
++ = extensive and +++ = very extensive tuberculosis involvement.
( ) = died during experiment.
R.I. ⊙ = regional inguinal nodes.
T.B. ⊙ = tracheobronchial nodes.

All injections were contained in a volume of 0.2 ml. Streptomycin was dissolved and diluted in sterile distilled water. PPD tablets of 0.05 mg. strength were suspended in sterile phosphate buffer solutions of pH 7.4.

All animals which survived were sacrificed eight weeks after infection. At necropsy the degrees of gross involvement of the local lesions, regional inguinal and tracheobronchial nodes, the spleen, liver, and lungs were noted.

The diet of all animals consisted of calf grower meal, carrots, and lettuce, (about 40 Gm. of the latter per guinea pig per day); a large bowl of cool tap water was provided for each cage for a period of two hours every morning around 8:00 A.M. The injections were given at 10:00 A.M. Lettuce and carrots were served in late afternoon; calf grower meal, consisting mainly of mixed cereals, was available at all times except during the drinking period early in the morning. The temperature in the animal house was kept at about 72° F.

## RESULTS

A quantitative grading of the macroscopic tuberculous disease in each animal of the four groups has been listed in Table I.

*A. Control Animals.*—All but two of the fourteen control animals had generalized tuberculosis. Two animals died of generalized tuberculosis during the experiment. Towards the end of the experiment the majority were beginning to lose weight (Fig. 1).

A tuberculin skin test performed one week before these animals were sacrificed showed a moderately intense reaction to 0.0001 mg. of PPD. Four animals showed the hemorrhagic necrotic type of reaction (Table II).

*B. Tuberculin Treated Animals.*—Thirteen of fourteen guinea pigs treated with tuberculin alone had generalized tuberculosis. Five guinea pigs in this group died with generalized disease during the experiment. Most animals showed a significant loss in weight during the last two weeks of the experiment. The skin reaction varied greatly, especially during the last three weeks. It was absent frequently; less frequently it showed hemorrhagic necrosis or the erythematous type of reaction (Table III).

*C. Streptomycin Treated Animals.*—In this group, the local lesions, when present, were small. The regional inguinal lymph nodes were usually small, minimally involved; the tracheobronchial nodes to a lesser degree. The spleen

TABLE II. TUBERCULIN REACTION IN GUINEA PIGS TREATED WITH STREPTOMYCIN ALONE AND IN CONTROL UNTREATED ANIMALS

| | REACTION NOTED IN INCREASING ORDER OF INTENSITY | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| NO. OF ANIMALS | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
| Streptomycin | – | – | – | – | – | – | ± | ± | ± | ± | ± | ± | + | + |
| Controls | ⊙ | ⊙ | ± | ± | + | + | + | + | + | + | (+) | (+) | (+) | (+) |

Test dose: 0.0001 mg.; animals tested seven weeks following infection.
⊙ 2 animals died some days before they could be tuberculin tested.
– = no reaction.
± = area of erythema less than 10 mm. in diameter.
+ = area of erythema 10 mm. and more in diameter.
(+) = area of necrosis or hemorrhagic necrosis.

was moderately diseased in three animals, slightly in six, and free of gross lesions in five. The liver was extensively affected in one animal, moderately in another, slightly in four, and free of gross lesions in eight. The lungs were grossly free in ten animals, moderately involved in two, and slightly in two. A constant gain in weight was observed in animals of this group.

Tuberculin skin tests were performed on these animals a week before they were sacrificed. Eight reacted mildly, six failed to react to 0.0001 mg. of PPD (Table II).

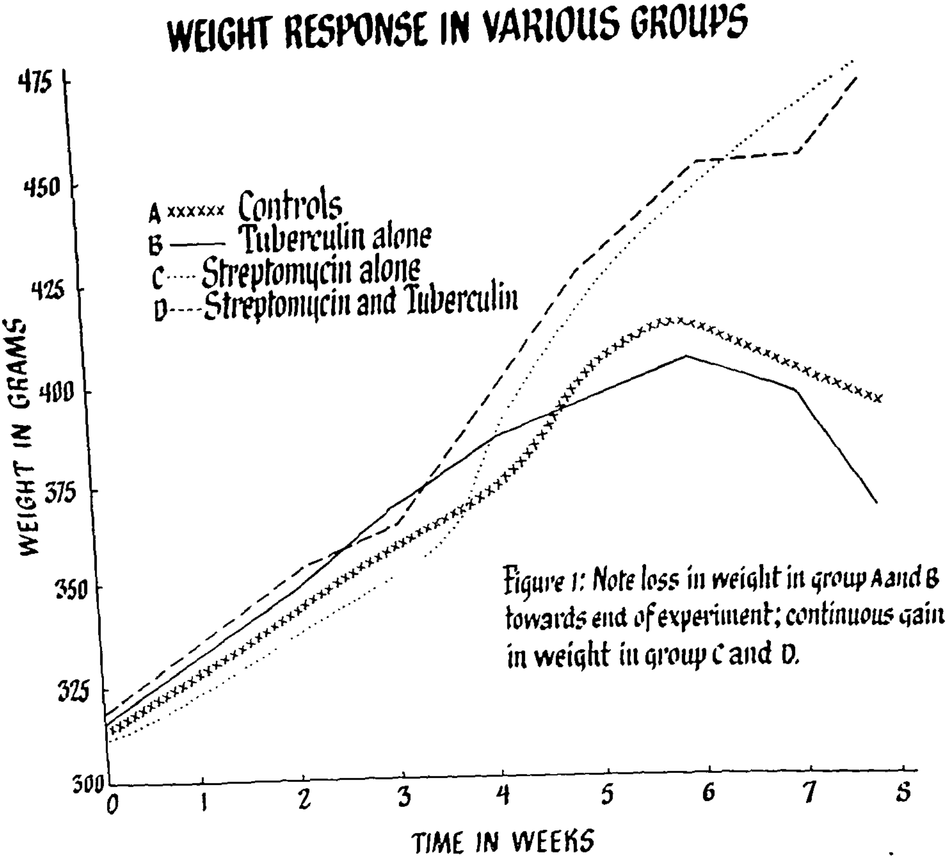

Fig. 1.

*D. Streptomycin and Tuberculin Treated Animals.*—Eight animals treated with streptomycin and tuberculin failed to show gross lesions. In six, the degree of macroscopic tuberculosis was slight. The findings in the local lesions, regional and tracheobronchial lymph nodes were similar to those noted in the streptomycin treated group. Constant gain in weight was observed. A positive skin reaction of slight to moderate intensity was generally noted in these animals following treatment (Table IV).

TABLE III. DEGREE OF TUBERCULIN REACTION IN GUINEA PIGS TREATED WITH PPD ALONE

| DOSE OF PPD (MG.) | DAYS AFTER INFECTION | REACTION NOTED IN INCREASING ORDER OF INTENSITY | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
| 0.00001 | 14 | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| 0.0001 | 18 | - | - | - | ± | ± | ± | + | + | + | + | + | + | + | + |
| 0.0002 | 21 | - | - | - | ± | ± | + | + | + | + | + | + | + | + | + |
| 0.0002 | 24 | - | - | - | - | - | - | - | - | ± | ± | ± | + | + | + |
| 0.0005 | 27 | - | - | - | - | ± | ± | ± | ± | + | + | + | + | + | + |
| 0.0005 | 31 | - | - | - | - | - | - | ± | ± | ± | ± | + | + | + | + |
| 0.0005 | 35 | - | - | - | - | ± | ± | ± | ± | (±) | + | + | (+) | (+) | (+) |
| 0.0002 | 38 | - | - | - | - | - | - | - | - | - | - | - | - | ± | ± |
| 0.0005 | 42 | ⊙ | - | - | - | - | - | - | - | - | ± | (±) | (+) | (+) | (+) |
| 0.001 | 45 | ⊙ | ⊙ | - | - | - | - | - | - | - | ± | ± | (+) | (+) | (+) |
| 0.001 | 49 | ⊙ | ⊙ | ⊙ | ⊙ | ⊙ | ± | ± | + | (+) | (+) | (+) | (+) | (+) | (+) |
| 0.0005 | 52 | ⊙ | ⊙ | ⊙ | ⊙ | ⊙ | - | - | - | - | - | ± | ± | + | (+) |

- = no reaction.
± = area of erythema less than 10 mm. in diameter.
+ = area of erythema 10 mm. and more in diameter.
(+) = area of necrosis or hemorrhagic necrosis.
⊙ = died.

TABLE IV. INTENSITY OF TUBERCULIN REACTION IN GUINEA PIGS TREATED WITH STREPTOMYCIN AND PPD

| DOSE OF PPD (MG.) | DAYS AFTER INFECTION | REACTION NOTED IN INCREASING ORDER OF INTENSITY | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
| 0.00001 | 14 | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| 0.0001 | 18 | - | - | - | - | ± | ± | + | + | + | + | + | + | + | + |
| 0.0002 | 21 | - | - | ± | ± | + | + | + | + | + | + | + | + | + | + |
| 0.0002 | 24 | - | - | - | - | - | - | - | ± | ± | ± | + | + | + | + |
| 0.0005 | 27 | ± | ± | ± | + | + | + | + | + | + | + | + | + | + | + |
| 0.0005 | 31 | ± | ± | ± | ± | ± | + | + | + | + | + | + | + | + | + |
| 0.0005 | 35 | ± | ± | ± | ± | + | + | + | + | + | + | + | + | + | (+) |
| 0.0002 | 38 | - | - | ± | ± | ± | ± | ± | + | + | + | + | + | + | + |
| 0.0005 | 42 | - | ± | ± | ± | ± | ± | ± | ± | ± | + | + | + | + | + |
| 0.001 | 45 | - | ± | ± | ± | ± | ± | ± | ± | ± | + | + | + | + | + |
| 0.001 | 49 | ± | ± | ± | ± | + | + | + | + | + | + | + | + | (+) | (+) |
| 0.0005 | 52 | - | ± | ± | ± | ± | ± | + | + | + | + | + | + | + | + |

- = no reaction.
± = area of erythema less than 10 mm. in diameter.
+ = area of erythema 10 mm. and more in diameter.
(+) = area of necrosis or hemorrhagic necrosis.
(=) Treatment with Streptomycin started fourteen days after infection; dose of 5 mg. daily for six weeks.

## DISCUSSION

Control animals and those treated with tuberculin alone, with but few exceptions, showed similar generalized types of disease consistent with a moderately large infective dose of tubercle bacilli.

Animals treated with tuberculin alone showed a higher mortality and a greater loss in weight than did the controls. It appeared that under the conditions of our experiment, treatment with PPD alone proved harmful to tuberculous guinea pigs.

Our findings in animals treated with streptomycin alone correspond to those reported by other workers. The organs of many guinea pigs revealed tuberculosis in the gross; but these lesions were of a minimal nature when compared with the extensive disease found in either the untreated controls or the tuberculin treated animals.

Animals treated with both streptomycin and tuberculin showed distinctly less tuberculosis than did those treated with streptomycin alone.

Independent objective microscopic study and grading of the tissues of streptomycin and streptomycin plus tuberculin treated animals revealed that those animals which received combined therapy developed significantly less tuberculosis than did the animals which received streptomycin alone. The spleens of the streptomycin treated guinea pigs showed more tubercles and slightly more caseation than did the spleens of streptomycin and PPD treated guinea pigs. The livers of streptomycin treated animals also showed more changes. Tubercle bacilli were demonstrated in some of the livers of streptomycin treated animals but none were observed in the streptomycin and PPD treated animals. The inflammatory reaction in the lungs of the streptomycin treated animals was more marked than it was in the lungs of the streptomycin and PPD treated animals.

The failure of a number of guinea pigs treated with streptomycin alone to react to tuberculin was first noted by Feldman and co-workers.[2] Six of our streptomycin treated animals did not react to 0.0001 mg. of PPD, while the remaining eight animals reacted only mildly when tested seven weeks after infection. The majority of our control untreated guinea pigs, also tested once on the same day with a similar dose of PPD, gave strongly positive skin reactions. Four exhibited hemorrhagic necrotic reactions.

In the group of fourteen guinea pigs treated with PPD only, a hemorrhagic necrotic reaction was a not infrequent finding during the last three weeks of the experiment. The same animal might develop two and even three such reactions at different sites upon successive testing at intervals of a few days. The erythematous type of reaction occurred irregularly during this period. In this group a complete lack of skin sensitivity was observed very often. The two extremes of no reaction or intense reaction on testing with a given dose on the same day was frequently noted. (See Table III for treatment doses.) A tendency towards tuberculin desensitization may have developed in some of these guinea pigs. Treatment with PPD probably hastened the death of some animals in this group. Loss in weight was greater in these animals than in the untreated controls.

The tuberculin reaction in the streptomycin-PPD treated group, in general, was positive and constant during the last four weeks of treatment. Progress towards desensitization was not apparent in these animals. Thus the type and extent of skin response elicited by 0.0005 mg. on the twenty-seventh or thirty-first day following infection was practically the same as that obtained with the same dose on the fifty-second day. (See Table IV for treatment doses.) Of 168 treatment doses given to guinea pigs of this group, in only three instances could the local intracutaneous skin reaction be classified as more than moderately erythematous; necrosis occurred twice and hemorrhagic necrosis once.

## SUMMARY

Guinea pigs infected with 0.01 mg. of tubercle bacilli (human strain H37rv), were treated with either tuberculin, streptomycin, or a combination of streptomycin and tuberculin (PPD).

Under the conditions of our experiments, treatment with tuberculin (PPD) alone proved harmful.

Guinea pigs treated with streptomycin plus tuberculin developed significantly less tuberculosis than those treated with streptomycin alone.

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2. Feldman, W. H., Hinshaw, H. C., and Mann, F. C.: Streptomycin in Experimental Tuberculosis, Am. Rev. Tuberc. 52: 269, 1945.

# SIMPLE CARDIAC DEFIBRILLATOR[*]

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A SIMPLE portable and inexpensive defibrillator, essential in any operating room, can be built for less than ten dollars of material. The parts required are: one ammeter 0 to 5 amperes, one fixed resistor 50 watts, one variable resistor 50 watts, and one ordinary push-button switch. The shocking electrodes for the heart are copper plates 3½ inches in diameter set in "plexiglass" plastic handles[†] 9½ inches long and ¾ inch in diameter.[‡] The copper plates can be covered with a fine chamois felt, if desired, to avoid overheating the heart.

The housing for the apparatus, less electric cords and electrodes, weighs 1 pound, 14 ounces (galvanized iron with plastic top). Its dimensions are: length 7 inches, width 4½ inches, height 3¼ inches.

The defibrillator utilizes ordinary 110 volt alternating house current, and is designed for use in cases of ventricular fibrillation associated with cardiac arrest occurring in the operating room. Its general construction and specific use has been described by Beck.[§] The chest may already be open for a thoracic operation. If not, an incision up to 10 inches, extending laterally from the sternum in the fourth or fifth interspace, is *quickly* made by the surgeon.

*Beck states that when there is no palpable pulse, no blood pressure, and no respiration, no time should be wasted. Cardiac resuscitation must be started at once.* He lists the following "*DON'TS*" if certain failure is to be avoided:

1. *Don't* listen for a faint heart sound.
2. *Don't* wait for an electrocardiogram.
3. *Don't* inject epinephrine through the chest wall into the heart.
4. *Don't* dilate the rectal sphincter.
5. *Don't* give mechanical respiration by compression of the chest.
6. *Don't* give a blood transfusion.
7. *Don't* give an intra-arterial transfusion.

Beck states that the surgeon and anesthetist have only three to five minutes to expose the heart and, by cardiac massage and oxygenation of the lungs, prevent irreversible cerebral anemia. After the interspace is incised, the fourth and fifth

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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[*] Developed in the Engineering Division, Veterans Administration Hospital, Amarillo, Texas, by Mr. George A. Welch, Engineer Officer, Mr. Leland B. Roper, and Mr. Fred H. Kelly, in accordance with the general instructions of TB 10-65, July 18, 1950.

[†] The handles are conveniently constructed from two halves cemented together around the wiring.

[‡] A set of children's electrodes, smaller in diameter, should also be available.

[§] Beck, C. S. Treatment of Cardiac Arrest. Veterans Administration Technical Bulletin, TB 10-65, July 18, 1950.

cartilages are cut, the wound retracted with a self-retaining retractor, and the heart massaged at a rate of 60 to 70 per minute. The surgeon determines the most effective method of massage. The heart can be rhythmically compressed against the sternum with one hand; or it can be compressed between the palm and fingers of one hand posteriorly and the fingers of the other hand anteriorly. It may be necessary to incise widely the pericardium if the latter mechanically interferes with effective cardiac compression. If rhythmic heart contraction is not restored in a reasonable time, ordinarily a few minutes, the pericardium must now be incised if this has not been done previously, to observe whether the heart is in asystole or fibrillation.

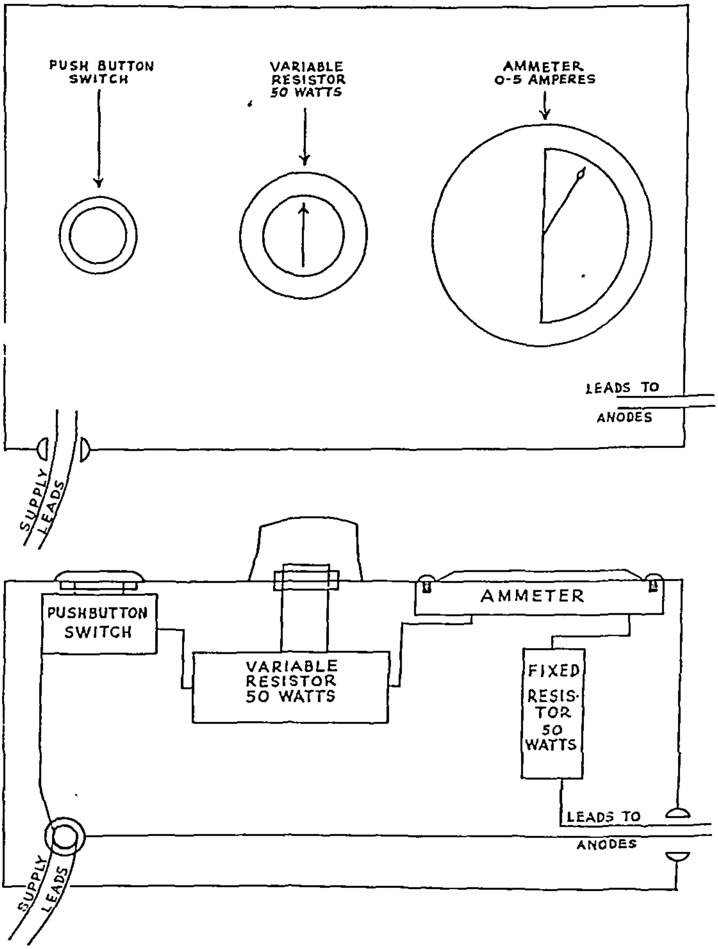

Fig. 1.—*Top*, plan of defibrillator panel as shown, left to right, push-button switch, variable resistor knob, and ammeter. *Bottom*, side view of parts wired in series.

If the heart is in ventricular asystole, injection of 5 to 10 c.c. of 1:10,000 epinephrine into the cavity of the right auricle or ventricle, plus massage, may restore the heart beat. Up to five injections of epinephrine can be given at intervals of three to five minutes (Beck). Failure to restore the beat indicates faulty lung aeration, faulty heart massage, or heart disease. Epinephrine increases heart tone, but is generally considered to predispose to ventricular fibrillation.

If, on the other hand, the heart is already in or should go into ventricular fibrillation, procaine is used to reduce the irritability of the heart. Procaine reduces heart tone, and therefore not over 5 c.c. of 1 or 2 per cent solution is used per injection. This amount of procaine is injected into the cavity of the right auricle or ventricle, and the heart massaged in order to distribute the drug through the coronary circulation. The defibrillator electrodes are then placed in contact with each side of the heart, as far apart as possible, and by means of a quick flip of the push-button switch, 1 to 1.5 amperes of current are sent through the heart for approximately one-fifth of a second. During this brief period of electric shock the muscle fibers are contracted. When the finger is released from the push button and the current broken, the ventricles may be in asystole or may again fibrillate. If asystole is present, epinephrine and massage are used as described above. If ventricular fibrillation is present, procaine plus massage are used as described above. If fibrillation persists after two electric shocks, no

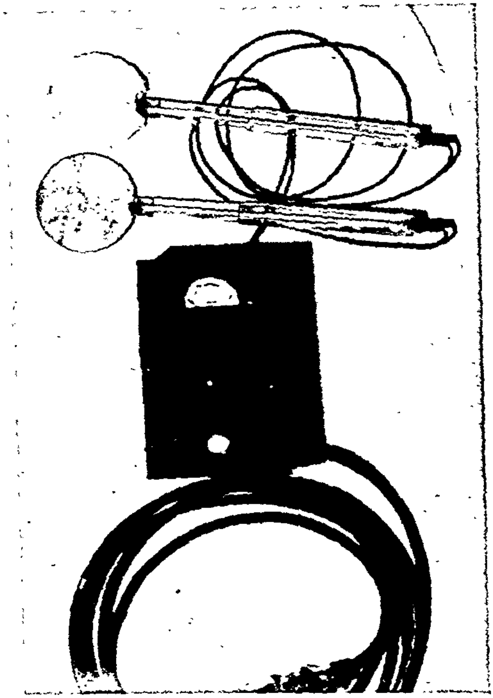

Fig. 2.—Top view of assembled defibrillator. The cords to plastic handle plug in and out for convenience.

From *top* to *bottom*, ammeter, knob for variable resistor, push-button switch. Apparatus delivers a *flow* of 1 to 1.5 amperes of current, and uses 110 *volt* alternating current. A quick flip of the finger on the push button will insure current flow for only the required ⅕ to ⅒ of a second.

more procaine is used, but massage is continued and shock may be tried a few more times. Beck states that in his experience epinephrine, although usually contraindicated in fibrillation, can still be cautiously administered in the presence of persistent ventricular fibrillation; it can "supply the spark that starts the beat after the next shock."

At all times—before, during, and after resuscitation of the heart—ample oxygenation of the lungs must be insured by adequate rhythmic inflation of the lungs with oxygen, through an intratracheal tube inserted at the earliest possible moment. *Rhythmic adequate inflation and deflation* of the lungs is paramount to insure proper oxygenation of the blood and release of carbon dioxide out of the blood. In a closed system, carbon dioxide is absorbed by fresh soda lime.

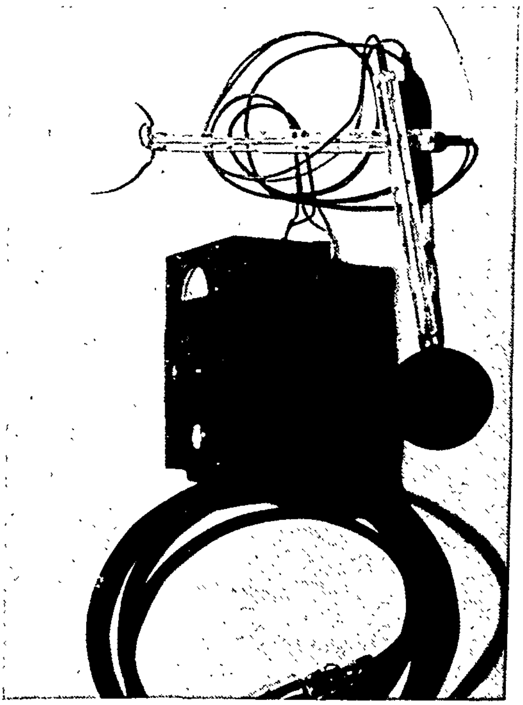

Fig. 3.—Oblique view of assembled defibrillator.

It may take 1 to 2 hours to restore the heart beat; the heart muscle must look pink throughout this period. A mechanical resuscitator is advisable for prolonged pulmonary ventilation; for shorter periods rhythmic compression of the anesthetic bag is usually practiced.

After cardiac resuscitation is achieved the pericardium is loosely closed with sutures, the chest wall tightly closed and the pneumothorax removed by suction through a catheter. Intravenous fluids may now be given for falling blood pressure. Mechanical resuscitation is given until spontaneous respiration is restored. The intratracheal tube is left in place until adequate spontaneous respiration is assured.

Beck's adage *"The beat can be restored in any normal heart"* should encourage the surgeon and anesthetist and discourage despair.

## ADDENDA

Since the preparation of this article, the writer has become aware that some have advocated the insertion in the defibrillation unit of an isolation transformer. While this may reduce to some extent the hazard of electric shock to personnel working near the patient, it will not eliminate it. The chief feature of the isolation transformer is to reduce the incidence of fuse blowing. Care to avoid contact of the copper electrodes is necessary to avoid blowing a fuse. Surgeons, anesthetists or other personnel who are or may come into contact with the patient or operating table should wear rubber gloves (preferably two pairs as a precaution against accidental perforation) and avoid grounding themselves by standing on a wooden platform or other nonconductor.

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Kay and Blalock found 10 per cent calcium chloride solution lifesaving in the treatment of spontaneous or postdefibrillation cardiac standstill. During operations for congenital heart disease in children, where 0.5 c.c. of 1 to 1,000 epinephrine injected into the left ventricular cavity failed to restore the heart beat, 2 to 4 c.c. of 10 per cent calcium chloride injected into this chamber produced dramatic restoration. Injection into the left ventricular cavity, plus massage, insures the passage of calcium chloride into the coronary arteries and is the preferred method. However, if the left ventricle is inaccessible, any other heart chamber may be used.

Calcium chloride is also valuable to *prevent* ventricular standstill in a heart with weak contractions. The calcium ion appears to act on the ventricular muscle, increasing its excitability and contractility. This effect resembles the accelerator stimulation of epinephrine and the muscular stimulation of digitalis. Calcium chloride solution should therefore be ready and available as part of the resuscitation armamentarium, in the event that epinephrine is ineffectual.

(Kay, J. H., and Blalock, A.: "The Use of Calcium Chloride in the Treatment of Cardiac Arrest in Patients," *Surg., Gynec. & Obst.* 93: 97, 1951.)

# SURGICAL TREATMENT OF MULTIPLE ARTERIOVENOUS FISTULAS IN THE RIGHT LUNG IN A PATIENT HAVING UNDERGONE A LEFT PNEUMONECTOMY SEVEN YEARS EARLIER FOR THE SAME DISEASE

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ARTERIOVENOUS fistulas of the lung is a relatively rare condition that is being reported with increasing frequency. The case we are reporting here was reported originally in 1944[1] and was the fifth case in the literature at that time. By 1949, however, Yater and associates[2] were able to tabulate forty-three cases, and added two new ones. Since that time reports of several more of these cases have appeared in the literature. An excellent review of the literature is given by Antonio Giampalmo.[3]

A 24-year-old white male welder was admitted to the University of Chicago Clinics on Jan. 24, 1943, complaining of frequent nosebleeds and colds for the last two or three years. He also had marked cyanosis with clubbing of the fingers and toes (Fig. 1, *A*). These findings were first noted shortly after an attack of influenza at the age of six years. Throughout 1941 and 1942 he had been inconvenienced by a mildly productive cough, which was aggravated when he was lying on the left side. He had worked as a farm laborer prior to 1942, and had never been incapacitated by his physical condition.

The family history showed that the patient's father had blue spots on his lips, and had been subject to numerous nosebleeds. Roentgenograms of the father's chest had revealed some shadows. Other male ancestors in the family were also reputed to have nosebleeds and blue spots on their lips. One of the patient's sisters died suddenly at an early age, and another one was said by the patient to be "blue," but she has not been seen by us.

At the time of admission in 1943, the red blood cells numbered 7,200,000 per cubic millimeter, and the hemoglobin content was 23 Gm. per hundred cubic centimeters. The arterial oxygen saturation was 71 per cent (van Slyke). Roentgenograms of the patient's chest (Fig. 1, *B*) showed a lobulated, moderately opaque area covering about 25 cm.$^2$ located between the left seventh and ninth ribs posteriorly. A second similar opacity 1 cm. in diameter was seen in the midaxillary line at the level of the right sixth rib. The clinical diagnosis was cavernous hemangioma of the left lung. As a result of this study it was thought at that time that the best treatment would be a left pneumonectomy. This operation was performed by one of us (W. E. A.) on Feb. 11, 1943. The pathologic diagnosis was multiple arteriovenous fistulas of the left lung (Fig. 1, *C* and *D*).

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From the Department of Surgery of the University of Chicago.

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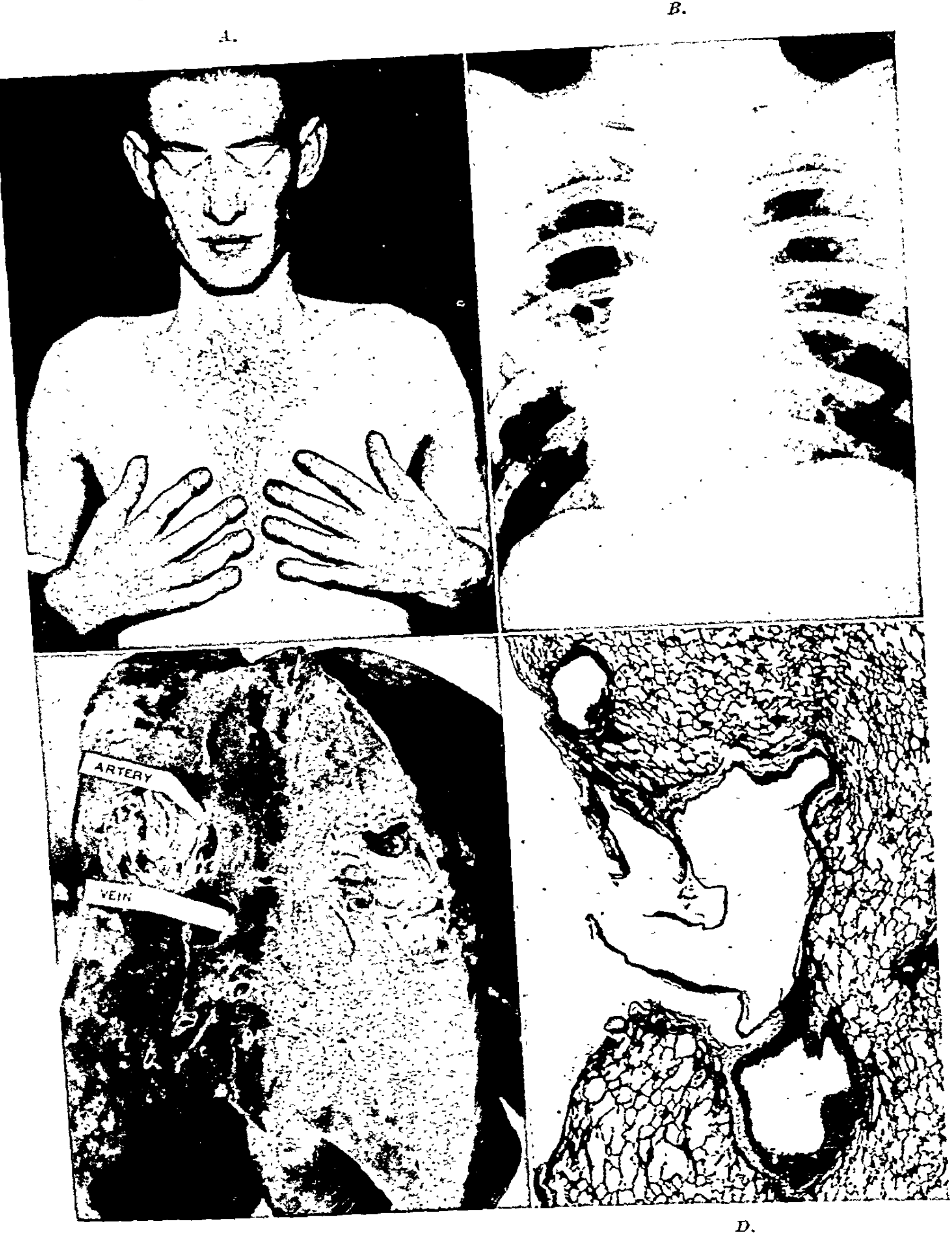

Fig. 1.—*A*, Patient before first operation; note marked clubbing of the fingers. (From Adams et al.: *Arch. Surg.*, 49: 51-58, 1944.) *B*, Roentgenogram before first operation, showing large irregular opacity in left lung and small (1 cm.) opacity near chest wall of right lung field. (From Adams et al.: *Arch. Surg.*, 49: 51-58, 1944.) *C*, Gross specimen of left lung; note large aneurysm with arterial and venous communications. (From Adams et al.: *Arch. Surg.*, 49: 51-58, 1944.) *D*, Microscopic section of portion of fistula. (From Adams et al.: *Arch. Surg.*, 49: 51-58, 1944.)

The patient's condition improved considerably following this surgery. His cyanosis largely disappeared. Upon discharge three weeks after operation his red blood count had fallen to 5,400,000, with a hemoglobin of 17.5 Gm. Two months later he started working again. Nine months following surgery his red blood count was 5,050,000, the hemoglobin 15.7 Gm.

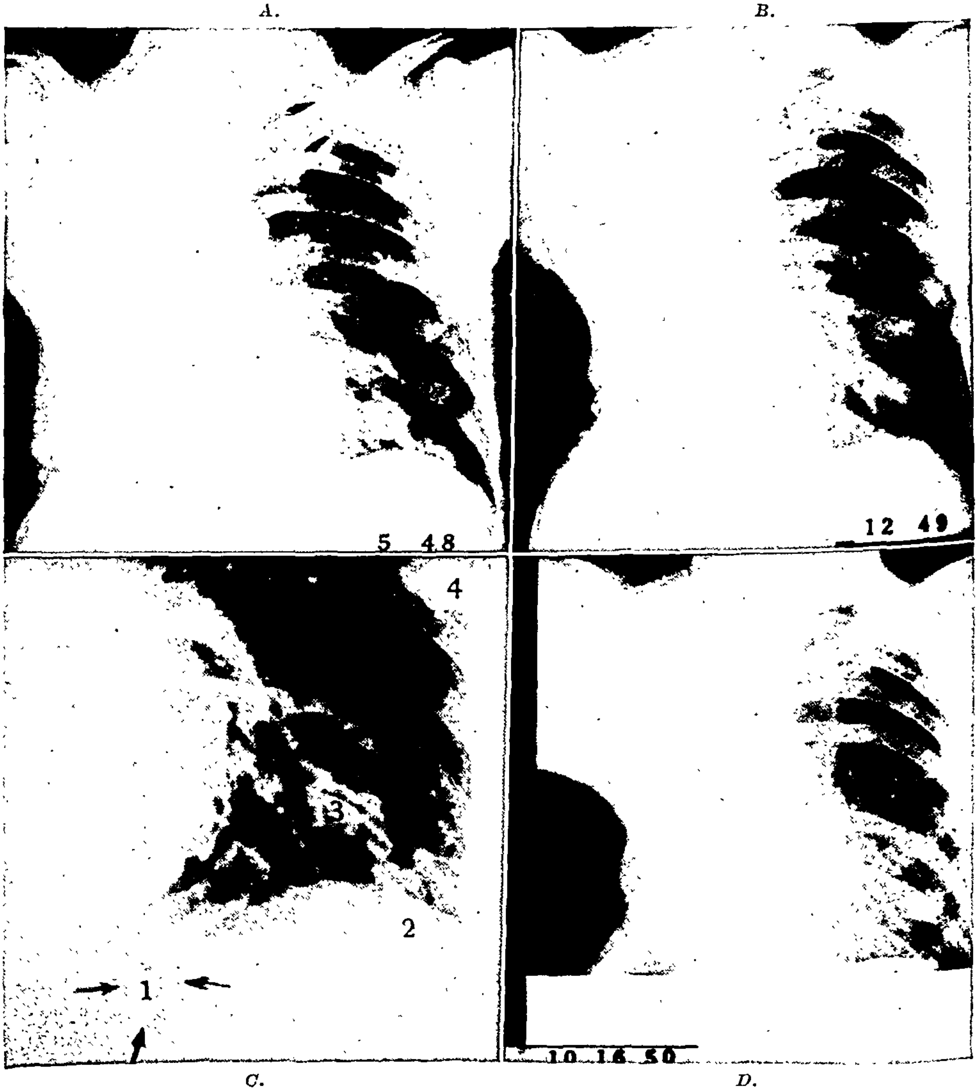

Fig. 2.—*A*, Roentgenogram after thoracoplasty. *B*, Roentgenogram just prior to second operation. *C*, Angiogram. Note the four fistulas filled with contrast material. *D*, Roentgenogram after last operation.

In 1945 a bronchopleural fistula with empyema was diagnosed; however, the patient did not return again until 1948. He then underwent a left thoracoplasty for his empyema. After this operation the patient felt well and continued to work as a farmer and welder. The red count at this time, however, had risen to 5,900,000, with a hemoglobin of 21 Gm. A chest roentgenogram (Fig. 2, *A* and *B*) showed some increase in the size of the right pulmonary lesion.

Fluoroscopy in 1949 showed the appearance of a new lesion in the lower right lung field. In October, 1949, pulmonary angiograms (Fig. 2, *C*) were made which revealed four cavernous spaces in the lower half of the right chest, the appearance of which was compatible with arteriovenous fistulas. Physical examination in February, 1950, revealed a well-developed, somewhat thin white man with marked cyanosis. Blood pressure was 125/80; pulse, 80; respiration, 22; temperature, 37° C. The lips showed some small hemangiomas. The left chest wall was partially collapsed. No bruit was heard over the right lung field. Except for the marked clubbing of the fingers and toes, no abnormalities were found in the extremities; urinalysis was negative. The blood count showed 7,900 white blood cells, with a normal differential. Hemoglobin was 22 Gm., the red blood cells 7,190,000, and the hematocrit 75 per cent. Arterial oxygen saturation was 69 per cent. Vital capacity was 1,100 cubic centimeter.

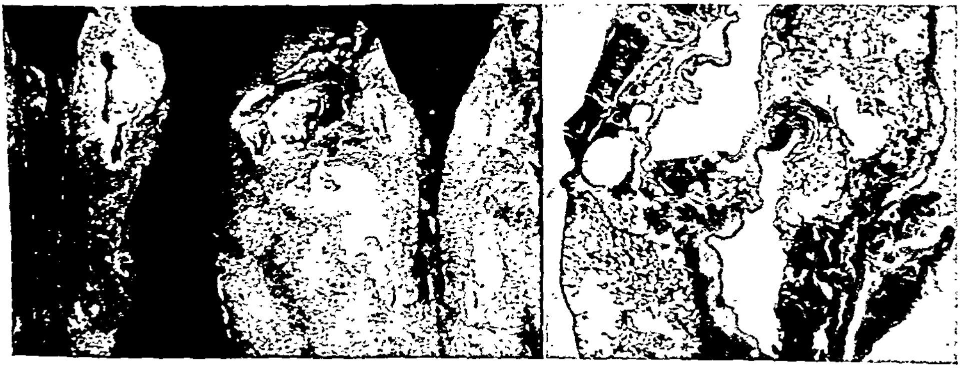

Fig. 3.—Gross and microscopic appearance of specimens removed at last operation.

Because it appeared clear that the patient was going steadily downhill, further surgery seemed necessary. Inasmuch as the lesions were located in each of the three lobes of the sole remaining (right) lung, the only possibility was to try to locate the fistulas and to ligate the efferent and afferent blood vessels, or to resect the pathologic areas, saving as much lung tissue as possible.

It was thought it would be wise to employ an oximeter during the operation, both as an aid for the anesthesiologist in evaluating the patient's condition, and as a possible help in identifying the fistulas, as one would expect an immediate rise in oxygen saturation with the compression of an area of the lung containing a fistula.

In order to test the validity of this reasoning, and to measure the degree of change which might be expected at the operation, an experiment was performed on a dog in which an end-to-end anastomosis was constructed between the pulmonary artery and pulmonary vein of the left lower lobe. A Millikan oximeter consisting of a Coleman anoxia photometer earpiece connected to a control box built by Dr. John Perkins, Jr., from our Physiology Department, according to Millikan's compensated circuit, was made into a recording instrument by leading the output to a Brown Electronik Potentiometer. In the experiment performed on the dog, the animal was heparinized and a glass cuvette containing a film of blood 1 mm. thick was connected in series with the cut ends of the femoral artery. The oximeter earpiece was applied to this cuvette.

As indicated in Fig. 4, occlusion of the anastomosis caused a 23 per cent rise in the per cent saturation of peripheral arterial blood, whereas release of occlusion caused a corresponding fall. This experiment, then, indicated that the Millikan oximeter would indeed be sensitive enough to distinguish between the normal pulmonary arteries on the one hand and the pulmonary artery-pulmonary vein shunts known to be present in the patient's lung. The oximeter was therefore used throughout the operation on the patient as indicated in the record (Fig. 5). The initial reading was set at 69 per cent, corresponding with the oxygen saturation found with the van Slyke method. When each one of three of the four shunts in the lung were occluded, the oximeter reading increased relatively abruptly by 7 or more per cent. At the end of the operation with still one fistula present and 100 per cent mask oxygen being administered the value was 90 per cent. Inasmuch as these three shunts consisting of cavernous hemangiomas lay on the surface of the lung and could be readily visualized, use of the oximeter in the manner described simply confirmed rather than established their location in this case. Nevertheless, we believe that in cases where shunts of this type lie deep in the lung, the method should prove valuable in locating them.

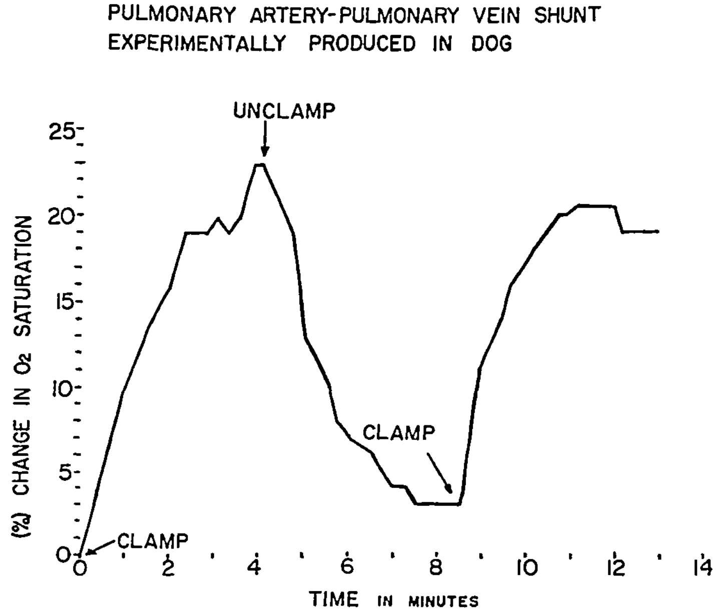

Fig. 4.—Oximeter tracing in a dog showing blood oxygen saturation with an arteriovenous fistula of the lung open and occluded.

A number of additional factors were considered prior to surgery, which seemed of considerable importance in this particular case. The pulmonary vascular bed had already been reduced by half by the previous pneumonectomy. It was thought that further reduction and consequent increased resistance by the occlusion of the large arteriovenous shunts, coupled with the abnormally high viscosity (found to be 2.3 times normal as measured with an Ostvald viscosimeter) of the blood because of the compensatory polycythemia, might impose too great a strain on the right ventricle.

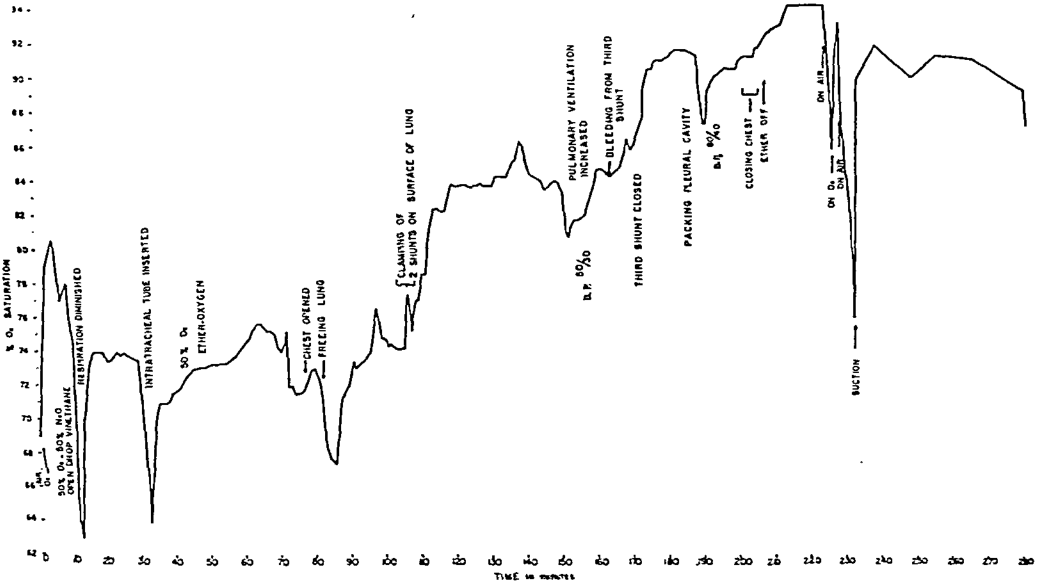

Fig. 5.—Oximeter tracing during operation; note sudden rise in oxygen saturation of the blood when shunts were occluded.

Plans were made to prevent this possible complication as follows: (1) right ventricular pressure would be measured throughout the operation in order to determine whether this pressure rose to dangerously high levels during ligation of the pulmonary shunts; (2) blood initially lost at operation would be replaced with saline, plasma or both, rather than with whole blood, in order to reduce the relative viscocity of the blood and thus minimize the rise to be expected during ligation. It was realized, however, that a fall in systemic blood pressure occurring in this patient at the time of ligation could be the result (1) of a sudden reduction in viscocity of the blood because of less hindrance by the peripheral vascular bed to the blood flow, (2) as well as of a possible reduction in right heart output resulting from the increased resistance in the pulmonary circuit following ligation of the shunts.

After these preliminary considerations, the operation was performed by one of us (W. E. A.) on Feb. 10, 1950. Under local anesthesia, a cardiac catheter was introduced in a jugular vein (as it was impossible to introduce it into an arm vein), and under fluoroscopic control placed in the right ventricle for pressure recordings (Table II). Intravenous normal saline solution was

TABLE I. BLOOD DATA. A SUMMARY OF THE CLINICAL COURSE AND LABORATORY FINDINGS IN A PATIENT WITH BILATERAL PULMONARY ARTERIOVENOUS FISTULAS

| DATE | THERAPY AND COMMENTS | VITAL CAPACITY | RED BLOOD CELLS | HEMOGLOBIN (GM.) | HEMATOCRIT | ARTERIAL OXYGEN SATURATION |
|---|---|---|---|---|---|---|
| 2/ 8/43 | | 3,800 | 7.2 | 23.0 | 82.0 | 71.0 |
| 2/11/43 | *Pneumonectomy, left* | | | | | |
| 2/15/43 | | | 6.4 | 17.0 | 51.5 | 72.0 |
| 3/ 4/43 | Discharged from hospital | | 5.4 | 17.5 | | |
| 11/22/43 | Working | 2,200 | 5.05 | 15.7 | 54.0 | 85.0 |
| 5/ 4/48 | Thoracoplasty | | 5.9 | 21.0 | | |
| 6/ 3/49 | New A-V fistula seen in right lower lobe | | 6.48 | 20.4 | | |
| 10/12/49 | | | | 24.0 | 70.0 | 78.0 |
| 2/ 3/50 | | 1,100 | 7.19 | 22.0 | 75.0 | 69.0 |
| 2/10/50 | *Excision of 2 and closure of one A-V fistula* | | | | | |
| 3/ 6/50 | | | 5.6 | 17.0 | 47.0 | |
| 4/ 7/50 | | 1,100 | 5.64 | 19.0 | 62.0 | 85.0 |
| 9/18/50 | | | 5.57 | 18.4 | | |
| 1/ 3/51 | | 1,400 | 5.35 | 17.4 | 59.0 | 86.0 |

started in both legs. Endotracheal ether-oxygen anesthesia was employed and, when the chest was opened, intermittent positive pressure was applied by assisted respiration throughout. Every fifteen minutes the hematocrit was measured. Blood loss was estimated by weighing sponges.

*Operation:* The right thorax was entered through the bed of the seventh rib, which was removed subperiosteally. The entire pleural space was found to be obliterated. Adhesions were freed over the right middle and lower lobes. One arteriovenous fistula was found at the surface of the lung at the lower medial aspect of the right lower lobe, and another was found at the lower lateral aspect of the right middle lobe. The fistulas were noted to pulsate, but no thrill was palpated. The two areas containing fistulas were clamped off with right angle clamps and resected. At about the time of closure of the second fistula the blood pressure dropped to 50/30. Transfusion with plasma

TABLE II. RIGHT VENTRICULAR PRESSURES DURING OPERATION, FEB. 10, 1950

| TIME | SYSTOLIC MM. HG | DIASTOLIC MM. HG |
|---|---|---|
| 9:20 A.M. | 40-50 | 20 |
| Induction period | | |
| 9:30 A.M. | 35-38 | 20-25 |
| 9:50 A.M. | 30-40 | 7-10 |
| 10:05 A.M. | 25-40 | 5-15 |
| 10:10 A.M. Chest open | 25-30 | 10 |
| 10:20 A.M. | 25-30 | 10 |
| 10:30 A.M. Vessels exposed | 25-35 | 10-15 |
| 10:40 A.M. | 25 | 10-15 |
| 10:50 A.M. 1st set of vessels clamped | | |
| 10:55 A.M. | 25-30 | 10 |
| 11:00 A.M. | 30 | 0– –2 |
| 11:15 A.M. 2nd set of vessels clamped | | |
| 11:20 A.M. | 30 | –2 |
| 11:40 A.M. 3rd set of vessels clamped | | |
| 11:45 A.M. | 40 | 0 |
| 12:00 M. | 45 | –2 |
| 12:25 P.M. | 45 | 0 |
| 12:30 P.M. Chest closed | | |

failed to produce a satisfactory rise in systemic pressure. Since the right ventricular pressure was *only* 30 mm. Hg (well within normal limits) it was decided to give whole blood. The systemic blood pressure rose to 80/40 soon after this transfusion. Freeing of the adhesions over the right upper lobe was next begun. As the posterolateral aspect of this lobe was approached, a thrill could be palpated.

*A third arteriovenous* fistula, located in the upper lobe, was broken into and bled considerably, causing a blood loss of 300 to 400 cubic centimeters. Clamps were placed proximal to control the bleeding. *This fistula was then* obliterated with multiple interrupted sutures. There appeared to be a fourth fistula deep in the substance of the lower lobe, but it was felt not *justified to* attempt its removal, because it would have been necessary to sacrifice too much lung tissue. Two Pezzar catheters were brought through the eighth interspace, and the wound was then closed in layers. The patient's condition remained relatively good throughout the procedure.

The hematocrit which was 75 per cent just before the operation had dropped to 60 per cent at the end of the procedure.

The estimated blood loss was 2,500 c.c. Replacement consisted of 1,200 c.c. of saline, 1,500 c.c. of plasma and 1,550 c.c. of blood, given during the operation which required three hours and fifteen minutes.

It can be seen in Table II that the systolic right ventricular pressure increased after clamping the fistulas, while the diastolic went down. The high right ventricular pressure during induction must be attributed to straining of the patient. As the viscosity came down gradually during the procedure, as indicated by the decrease in hematocrit, one might have expected a drop in the abnormally high right diastolic pressure recorded at the start.

The patient's postoperative course was quite stormy for the first week. He had much difficulty raising secretions, and bronchoscopy and intratracheal aspirations were needed rather frequently. He remained quite apprehensive, but was able to be up and about most of the day in a wheel chair after the first week. At the time of discharge (four weeks after the operation) he was walking around the ward. His vital capacity at that time appeared to be less than 1,000 cubic centimeters.

At the present time (January, 1951) the patient is in fairly good condition. At rest he is not cyanotic, and is able to do some light exercise. He can walk up two flights of stairs slowly without resting, walk several miles on the level, and is much less short of breath than before operation. More than slight exercise, however, is too much for him, for he then becomes rapidly short of breath and markedly cyanotic. His blood oxygen saturation in room air is 86 per cent and goes up to 100 per cent on inhalation of 100 per cent oxygen. His red blood count is 5,350,000; hematocrit, 59; and hemoglobin, 17.4 Gm. The vital capacity has increased and is now 1,400 c.c. In general the patient feels much better than prior to surgery. However, in view of the fact that he developed more arteriovenous fistulas after one operation, his prognosis is uncertain.

## DISCUSSION

It is generally accepted that the best treatment in these cases is to remove as little lung tissue as possible, since pre-existing fistulas may open up or new ones may develop following surgery and cause a recurrence of the clinical picture. In this case it was thought in 1943 that a pneumonectomy was the best treatment. It would, however, have been better to have performed a partial resection of the lung. Since the condition in most of these patients gradually gets worse, as indicated by the decreasing oxygen saturation and increasing polycythemia, surgery should always be considered. The prognosis without surgery is usually unfavorable, the cause of death being cerebral thrombosis, pulmonary bleeding, or heart failure.

## SUMMARY

A case of multiple arteriovenous fistulas of both lungs, for which parts of the right lung had to be resected seven years after a left pneumonectomy, is presented. Difficulties encountered are discussed. The patient improved considerably after surgery. However, in this patient the prognosis must remain guarded.

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# A USEFUL BRONCHIAL CLAMP

WALTER J. BURDETTE, PH.D., M.D.
NEW ORLEANS, LA.

A BRONCHIAL clamp* (Fig. 1) has been designed which has proved very useful in the management of the bronchus during pulmonary resection. It consists of thin blades with serriform jaws 3.5 cm. in length at right angles to a handle 20.7 cm. long containing a screw mechanism for closing the jaws. The two parts of each instrument may easily be disassembled for cleaning.

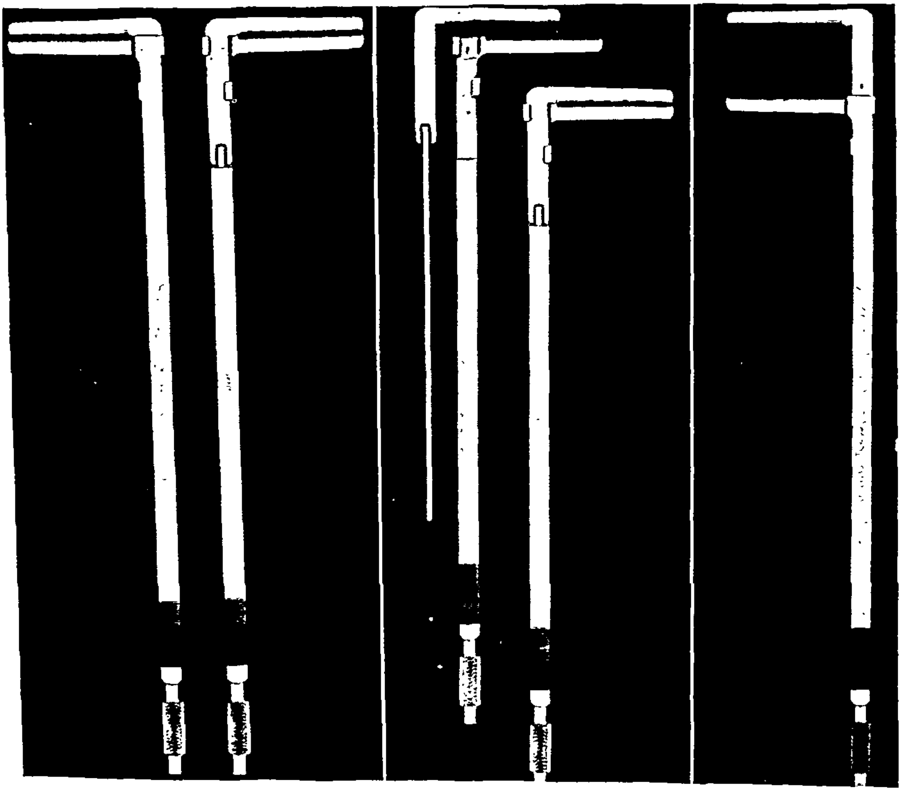

Fig. 1.—Bronchial clamp showing serriform jaws open, closed, and disassembled for cleaning.

A syringe fits the end of the handle and may be used to clean the bore. The instrument is constructed of stainless steel and is quite sturdy, although it weighs only 37 Gm. It is sterilized in oil and left with a light coating of oil when not in use.

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From the Department of Surgery, Louisiana State University School of Medicine.
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*Manufactured by Mr. B. Richter, 843 Duane Street, Glen Ellyn, Illinois.

The thin blades permit easy application, and the screw adjustment and serriform jaws prevent slipping, avoiding the necessity of crushing the tissue. The absence of ratchet handle and the construction of the shaft at right angles to the blades result in the clamp being placed at 90 degrees to the bronchus. This prevents the handle from lying in the axis of the bronchus where the distal pulmonary parenchyma interferes with proper use of the instrument. When the clamp is in place on the proximal bronchial stump it is useful in manipulating the stump as sutures are placed.

When the bronchus is closed on the proximal side without the use of a clamp, either before or after it is cut, paired clamps are not used, but a single clamp is useful in occluding the distal portion of the bronchus which is to be removed. This, of course, is desirable as early as possible to prevent contamination of the remaining tracheobronchial tree by the part to be resected. Although the instrument is primarily constructed for work with the bronchus, it has some usefulness in vascular surgery as well.

## SUMMARY

A stainless steel bronchial clamp has been designed with serriform blades and screw handle which is convenient to apply with little danger of slipping and minimal damage to tissue.

# GANGRENE OF THE ARM FOLLOWING USE OF THE SUBCLAVIAN ARTERY IN A PULMONOSYSTEMIC (BLALOCK) ANASTOMOSIS

WATTS R. WEBB, M.D., AND THOMAS H. BURFORD, M.D.
ST. LOUIS, MO.

THE occurrence of gangrene of an extremity following interruption of the subclavian artery incident to the performance of an anastomosis to the pulmonary artery has been an extremely rare complication. There has been only one such case previously reported,[4] in the many hundreds of anastomoses. This of course demonstrates the very efficient collateral circulation of the shoulder girdle. Very little, however, has been written about the exact patterns of this collateral. Our interest was stimulated by the following case.

## CASE REPORT

B. J. B., a 1-year-old girl, entered Children's Hospital on Feb. 6, 1950, because of cyanosis since birth. The parents noted that the child had dyspnea when eating or crying, and occasional attacks of syncope. She had had frequent upper respiratory infections with slow recovery. She had gained eight pounds since birth. Because of the poor progress shown by the child while under observation through the outpatient clinic, early operative intervention was deemed necessary.

Physical examination showed a small, cyanotic, moderately well-developed infant girl with clubbing of the fingers and toes. She demonstrated marked dyspnea when crying or eating. The tonsils were enlarged and injected. The lungs were clear. The heart appeared enlarged to percussion both to the right and to the left. A faint systolic murmur and thrill were present over the precordium, with greatest intensity in the third interspace to the left of the sternum. The blood pressure was 90/50, with equal pulses in all extremities.

The laboratory data showed a normal urine, a white blood cell count of 12,000 per cubic millimeter with a normal differential, and 7.2 million red blood cells with 15.2 Gm. hemoglobin. The electrocardiogram showed sinus tachycardia and right axis deviation, and was suggestive of myocardial damage. Chest roentgenograms showed findings typical of a tetralogy, with inconspicuous vascular shadows bilaterally and a concave pulmonary conus. There was moderate right ventricular hypertrophy, and the aortic window appeared clear. The aortic arch was on the right.

The patient improved on digitalis and oxygen, and the acute tonsillitis responded to penicillin and streptomycin therapy.

*Operative Findings.*—On Feb. 13, 1950, a left thoracotomy was performed through a posterolateral incision with resection of the fourth rib. The aorta was found to descend on the right side, as had been thought preoperatively. Though the subclavian and pulmonic vessels were very small, it was decided to attempt an anastomosis. In order to obtain sufficient length the subclavian artery was transected distal to its first four branches, and the internal mammary, vertebral, costocervical, and thyrocervical vessels were individually ligated. An end-to-side anastomosis of the subclavian to the pulmonary artery was performed. A thrill at the end of this procedure indicated its patency.

Postoperatively the patient showed only slight improvement in color, and a murmur typical of a shunt was never identified. On the first day after operation lividity and coolness of the left forearm were noted. Because these changes might be due to thromboses, inter-

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CHART I. MAJOR COLLATERAL CHANNELS AFTER SIMPLE LIGATION OF THE SECOND PART OF SUBCLAVIAN ARTERY

| | | | | |
|---|---|---|---|---|
| First portion of subclavian | Transverse scapular and Transverse cervical of the thyrocervical | with | Thoraco-acromial<br>Subscapular<br>Circumflex scapular | to axillary |
| First portion of subclavian | Internal mammary<br>Superior intercostals of the costo-cervical<br>Aortic intercostals | with | Pectorals of thoraco-acromial<br>Superior thoracic<br>Lateral thoracic<br>Subscapular<br>Circumflex scapular<br>Dorsal thoracic | to axillary |
| First portion of subclavian | Unnamed branches passing through axilla | with | Branches of the axillary | |

CHART II. COLLATERAL CIRCULATION AFTER LIGATION OF THE SECOND PORTION OF SUBCLAVIAN ARTERY AND LIGATION OF THE INTERNAL MAMMARY, VERTEBRAL, THYROCERVICAL, AND COSTOCERVICAL TRUNKS

| | | | | |
|---|---|---|---|---|
| Contralateral inferior thyroid<br>Homolateral superior thyroid | Inferior thyroid | Transverse scapular<br>Transverse cervical<br>Aortic intercostals | with | Thoraco-acromial<br>Subscapular<br>Circumflex scapular<br>Dorsal thoracic |
| Homolateral carotid muscular branches<br>Homolateral occipital<br>Contralateral vertebral muscular branches | Ascending cervical<br>Deep cervical | | | |
| Inferior epigastric<br>Aortic intercostals | Internal mammary<br>Superior intercostals | | with | Pectorals of throraco-acromial<br>Superior thoracic<br>Lateral thoracic<br>Subscapular<br>Circumflex scapular<br>Dorsal thoracic |
| Aortic intercostals | Unnamed branches passing through axilla from branches of subclavian | | with | Branches of the axillary |

mittent injections of intravenous heparin were started. However, adequate clotting time levels could not be consistently maintained. By the third postoperative day there was demarcation of the changes at the junction of the upper and middle thirds of the forearm. On the fifth postoperative day the patient developed right facial weakness and weakness of the right arm and leg, with increased tendon reflexes on the right apparently from a cerebral thrombosis. These symptoms gradually cleared, but the left forearm proceeded to dry gangrene and mummification. The patient's cyanosis returned to its preoperative intensity indicating the anastomosis had thrombosed. On March 23, 1950, the left forearm was amputated with primary healing of the wound. The patient was discharged three weeks later still showing cyanosis and having occasional episodes of syncope.

## DISCUSSION

Universally, after division of the subclavian artery, the arm becomes pulseless, and frequently cool. This gradually improves and, while only a faint radial pulse may return, in time the arm seems to become as well developed, as strong, and as warm as its fellow member. In attempting to explain the degree of ischemia seen above we were led to consider several factors, the first of which is the collateral circulation. After simple ligation of the second part of the subclavian the major collateral channels are as shown in Chart I. Thus, blood may leave the subclavian through the internal mammary, thyrocervical, and costocervical trunks and be efficiently shunted around the point of ligation. The vertebral contributes only a very few further collateral channels.

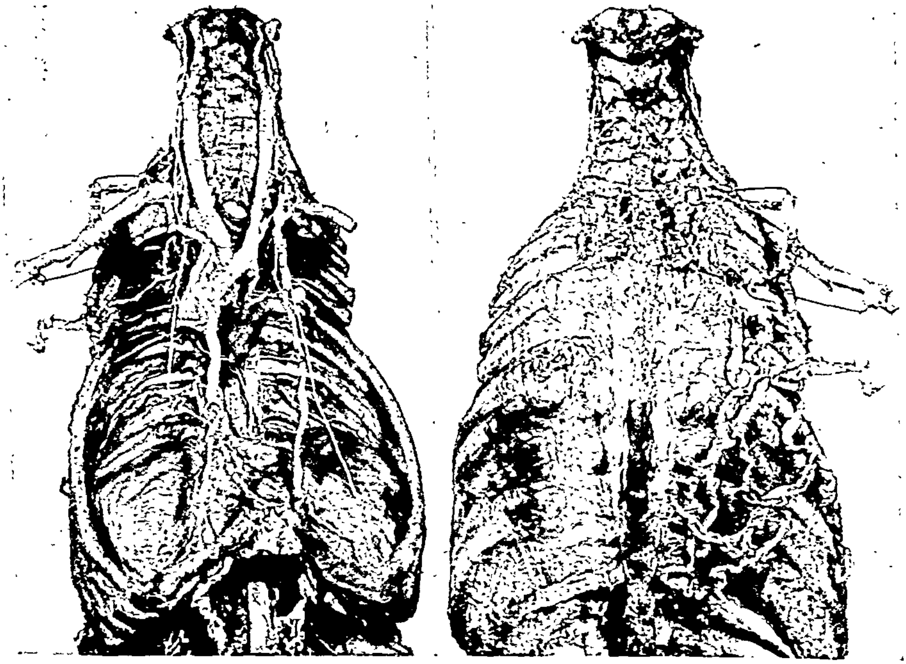

Fig. 1.—Anterior and posterior views of an injected specimen with coarctation of the aorta. This shows the prominence and multiplicity of the available intercostal and subscapular collateral pathways.

The specimen is that of a 65-year-old white man, prepared by Dr. E. E. Devereux and Dr. R. B. Stortz, from the Museum of the Department of Anatomy, Washington University School of Medicine.

This is in marked contrast to the circuitous route necessitated by ligation of the subclavian in its second portion when, in addition, its four trunks must be ligated at their origin (Chart II). In our experience this has seldom been necessary, but it has been done on other occasions without ill effect. In this case all blood passing through the channels depicted in Chart I must come from sources more distant than the first portion of the subclavian. The major routes in this situation are shown in Chart II and Fig. 1.

Of these many sources of blood flow the most important is the intercostals, principally from the posterior and lateral branches, with their anastomoses to the subscapular and the lateral thoracic vessels. This is visually very strikingly demonstrated by the injected specimen of a case of coarctation of the aorta (Fig. 2). Although the situations are not exactly analogous, the accompanying picture displays an exaggerated form of the usual anastomotic patterns.

Lam believed the posterolateral incision was responsible for the gangrene in the case he reported. He did not, however, give full details of the site of division of the subclavian artery, so that it is not possible to adjudge all the relative factors. It would seem that the posterolateral incision in comparison would interrupt more collateral circulation of the arm than would the anterolateral approach. The posterolateral incision divides branches of the subscapular and of the descending ramus of the transverse cervical arteries, and severs communication with the posterior and lateral divisions of all intercostals below the fourth or fifth intercostal space according to the site of the incision. The anterolateral incision divides the internal mammary, the supreme thorcacic, the thoracic branches of the thoraco-acromial trunk, and the anterior branches of the superior and aortic intercostals. Both types of incision divide the lateral thoracic artery with the anterolateral incision doing so at a higher level. The sum total seems to give some slight advantage to the anterolateral incision, but in view of the large number of Blalock procedures done through the posterolateral incision without difficulty even in older children and adults we are convinced that the site of incision is not too important.

*A far more significant factor, it seems to us, is that of thrombosis* of some portion of the collateral circulation. These individuals with their polycythemia have a well-recognized tendency to spontaneous intravascular thrombosis[8] which is accentuated postoperatively. Ligation of a mother trunk, for example, the thyrocervical, would enhance the possibility of local thrombosis with propagation into its diverging branches. That this factor played a role here is suggested by the evidence of thrombosis developing at the site of the subclavian pulmonary anastomosis and in the cerebral circulation.

Another possible factor is spasm of the arteries. The importance of this has been recognized by many workers, especially those dealing with traumatic vascular injuries, such as those incurred during war.[1, 7] The experiments of Kinmonth, Simeone, and Perlow[3] are particularly pertinent in pointing out the prompt appearance of arterial spasm after many types of mechanical trauma, such as operative manipulations. Usually the constriction is seen in only a localized segment of the traumatized vessel for a brief period of time, but it may persist for days, especially in the presence of nerve and other soft tissue injuries. Vasospasm is difficult to evaluate in any given case, and hard to recognize except by diagnostic anl therapeutic sympathetic nerve blocks. When, as in this instance, blocks have not been done, one can only speculate as to its importance.

The site of demarcation of the gangrene in this case is an interesting feature for which no exact explanation is at hand. While in the leg it is possible

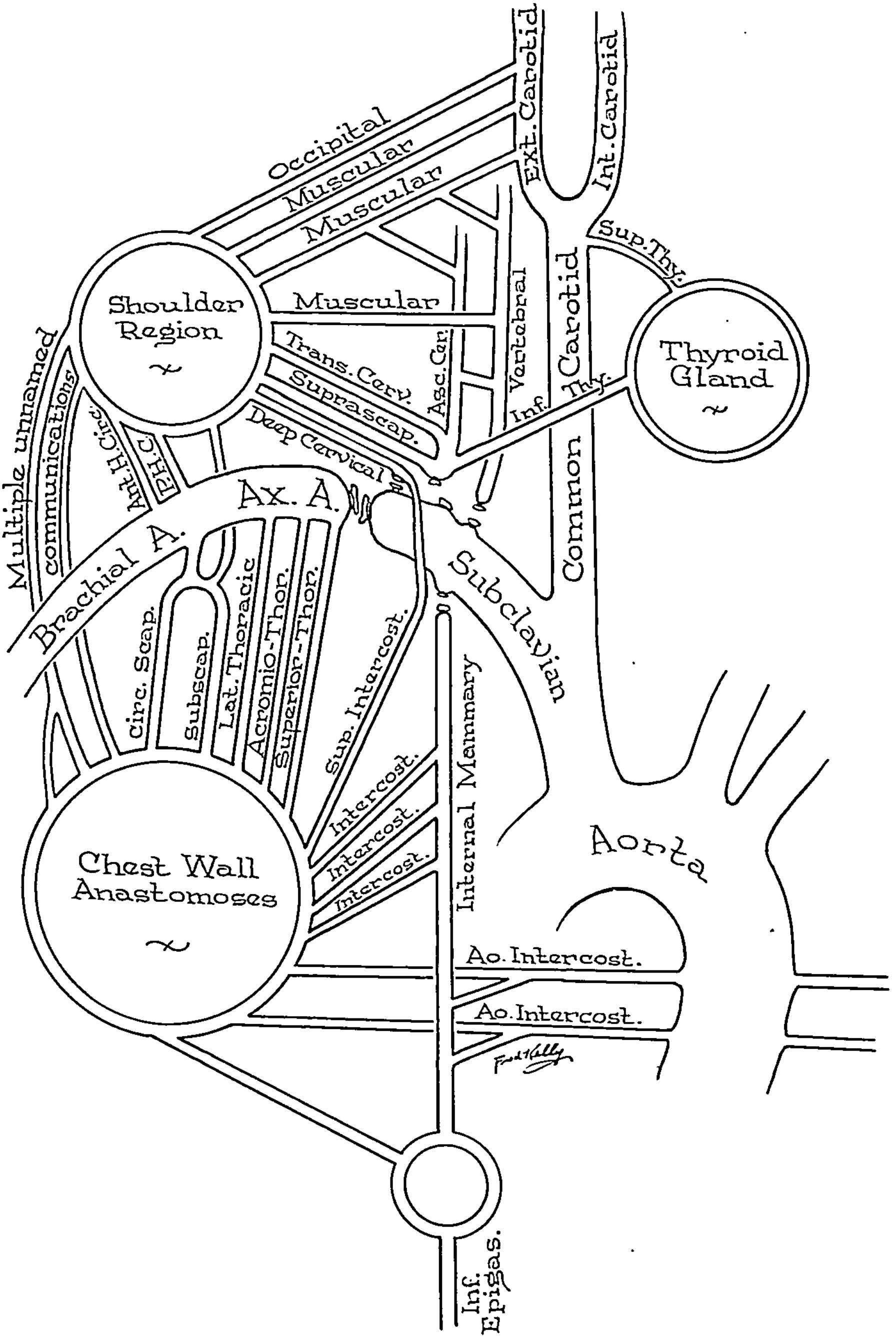

Fig. 2.—Diagram of the collateral circulation to the arm after ligation of the second portion of the subclavian artery and its branches.

to associate a given level of gangrene with arterial occlusion at a relatively definite point, this cannot be done in the arm. Since nearly all the data for gangrene developing after occlusion of the arteries of the axilla or arm have come from traumatic injuries when there have been associated injuries to the collateral circulation, they are not applicable to this problem.[2, 6] Leriche and Policard[5] found the common brachial artery proximal to the orifice of the profunda to be the only dangerous site for ligation. They report one case with subsequent gangrene of the digits of the hand and one case with atrophy and fibrosis of the upper arm and hand muscles. They state nothing, however, about the type of injury or the collateral circulation and collect thirty-one cases of ligation of the brachial artery at this point without subsequent gangrene. It is obvious that circulation to the distal parts is more seriously impaired than that of the more proximal portion of the extremity, and the demarcation must occur at some point along the gradient. The minimal vascular bed in this case undoubtedly lays in the collateral of the shoulder girdle at the sites of vasosection and thrombosis.

## SUMMARY

1. A case of gangrene of the arm following use of the subclavian artery in a Blalock type anastomosis for tetralogy of Fallot in a one-year-old child is presented.

2. The collateral circulation of the arm is discussed.

3. The effects of the type of incision, of intravascular thrombosis, and of vasospasm are reviewed.

We wish to express our appreciation to Dr. Mildred Trotter and Dr. Edward W. Dempsey of the Department of Anatomy, Washington University School of Medicine, for their kind cooperation and assistance.

## ADDENDUM

Since this was submitted for publication, one further case has come to our attention. F. D'Allaines in *Chirurgie Du Coeur*, Paris, 1950, Scientifique Francaise, reports the development of gangrene of the entire left arm of one patient after the subclavian artery had been anastomosed to the pulmonary artery. The operation was performed through an anterolateral incision, which we feel lends support to our view that the location of the incision is not an important factor. Details of the patient's age and of the level of transection of the subclavian artery are not given.

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# FIBROMA OF THE LUNG

## CASE REPORT

PAUL D. CRIMM, M.D., AND FREDERICK L. KIECHLE, M.D.
EVANSVILLE, IND.

PRIMARY benign tumors of the lung are not very frequent in occurrence. Undoubtedly, many of these take origin from the bronchus and can be considered as bronchial tumors. The larger tumors are those described usually as lung neoplasms, while the smaller ones are regarded as bronchial neoplasms. Benign tumors[1] include papilloma, fibroma, lipoma, myoma, chondroma, osteoma, and adenoma, which is considered by Graham and Womack[2] to be potentially malignant.

Among cases of fibroma of lung previously reported are those of Staffieri and associates,[3] and of Houyez.[4] Lindgren[5] reports that fibromas represent 9 per cent of the bronchial tumors reviewed by him. Lyssunkin[6] reports a fibroma of the lung which underwent malignant change to a xanthomatous fibrosarcoma. Lenk[7] mentions that sarcomas of the lung are quite rare, comprising an estimated 0.009 to 0.02 per cent of all autopsies.

Benign lung tumors may give no indication of their presence for years, until they attain such size that they cause pain, circulatory embarrassment, dyspnea, or pulmonary osteo-arthropathy. Blockade of bronchi, either by compression or tumor growth, gives rise to cough and inadequate drainage. These symptoms of obstruction often are diagnosed as bronchitis or pneumonia.

### CASE REPORT

This patient was a white man, aged 48 years, occupation laborer. He complained of persistent cough and pain in the right chest, which was present four weeks prior to consulting his local physician. Pain was of sharp, migratory, and intermittent character, beginning just inferior to the left costal border and passing in a narrow band up the fifth left interspace and then crossing to right chest at the same level. Cough, slightly productive from the onset, was first noticed about September, 1949. This cough appeared when he was reclining, and very seldom occurred in the sitting posture. Hemoptysis was absent. Right-sided chest pain was more of an aching character. There was a fifteen pound weight loss over a two-month period. No joint pains were noted. Past history was noncontributory. Family history revealed no evidence of tuberculous infection, nor of neoplastic disease.

*Physical examination* revealed the following positive findings: Nasal septum was deviated slightly to the right, but without obstruction. Thorax was asymmetrical, with respiratory movements slightly limited on the left side. Dullness and absence of breath sounds existed in the left base.

*Laboratory findings* were as follows: Urinary findings were essentially negative. WBC 10,300; basophils 3 per cent, eosinophils 2 per cent, juveniles 1 per cent, stabs 3 per cent, segmenters 71 per cent, lymphocytes 20 per cent. Sedimentation rate was 28 mm. (Cutler method). RBC 3,110,000, with 60 per cent Hb. (Sahli). Blood type "O." Bleeding time was four minutes, and clotting time was one and one-half minutes. Postoperative blood count

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From Boehne Tuberculosis Hospital.

Received for publication Aug. 29, 1951.

was: WBC 8,900; eosinophils 2 per cent, stabs 2 per cent, segmenters 73 per cent, lymphocytes 23 per cent; RBC 2,960,000, Hb. 54 per cent. Following transfusions, counts rose to RBC 3,280,000, Hb. 60 per cent; and RBC 4,170,000, Hb. 74 per cent.

Bronchogram (Fig. 1) in October, 1949, showed atelectasis in the left lower lobe and demonstrated obstruction of the lower bronchus, presumably by a tumor mass. The tumor was visualized at the time of bronchoscopy and biopsy obtained showed architecture of benign fibroma.

*Operative Report:* Tumor mass comprised most of the lower lobe, which was firmly adherent laterally to the chest wall. The inferior and medial adhesions were separated readily. The upper lobe was fixed by adhesions. The lower lobe and tumor were removed by the usual individual ligation technique. No sign of metastasis or extension of tumor within the operative field was observed. A lymph node near the hilus contained necrotic fluid. Several other slightly enlarged lymph nodes were removed. Recovery was uneventful. Patient was discharged on the sixteenth postoperative day with remaining lobe expanded.

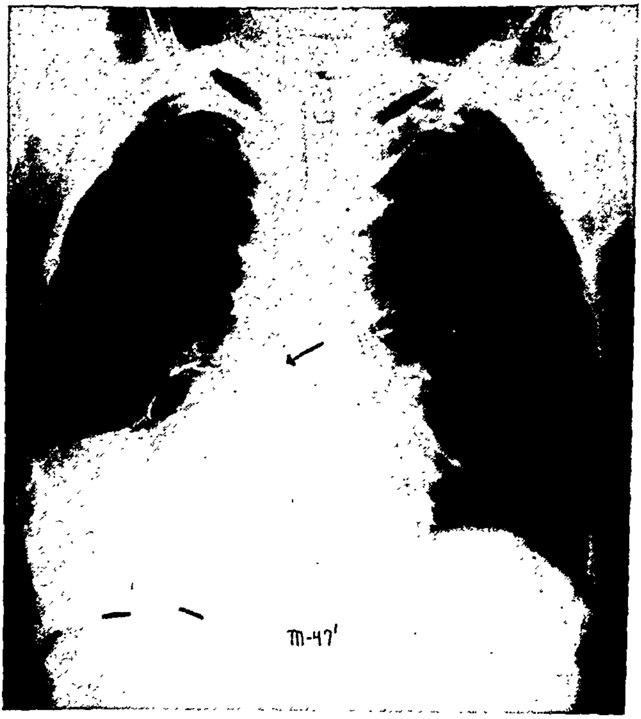

Fig. 1.—Bronchogram shows blockade of left lower bronchus and increased density throughout the lower lobe.

*Gross Pathology:* The tumor occupied more than half of the lower lobe volume. The weight of the lower lobe with the tumor was 740 grams. Hemisection (Fig. 2) demonstrated two spherical tumors, the one much larger and of firm rubbery consistency and suggestion of whorled pattern, with slightly pinkish-yellow color and areas of white and slightly softened degeneration; the other of smaller size, equally firm rubbery consistency, but with white glistening surface. Cut surfaces bulged. Extensions of tumor tissue were seen to fill three major bronchi, and to cross a bifurcation in such a manner as to suggest the arrangement of a staghorn calculus of the kidney pelvis. There was no evidence of invasive character, and the tumor appeared to be partially encapsulated, and was well demarcated from surrounding tissues.

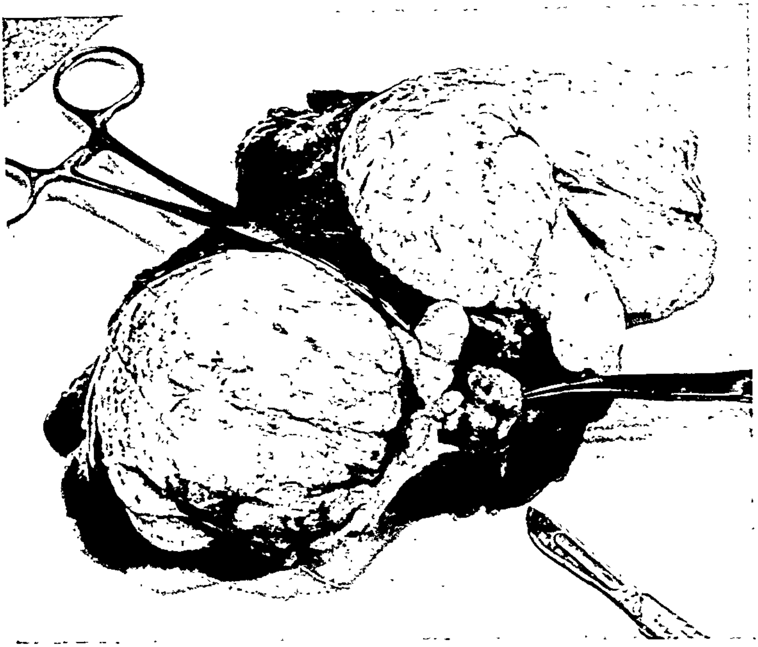

Fig. 2.—Hemisection of lower lobe shows character of tumor mass with extension into large bronchi.

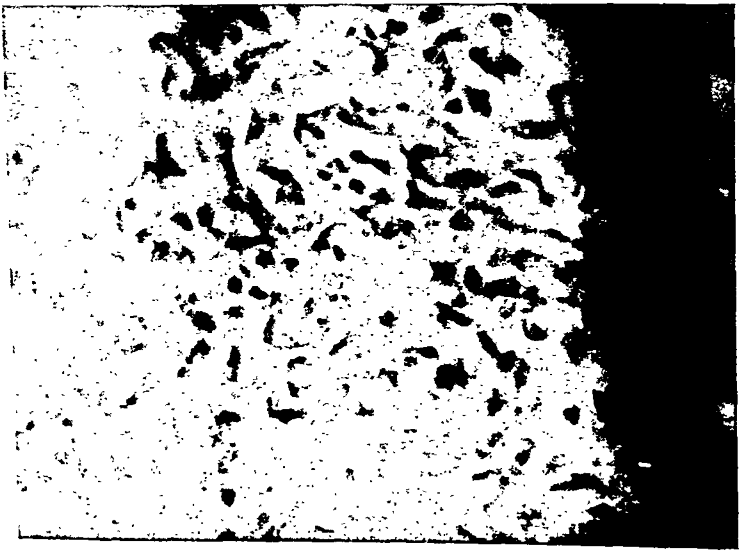

Fig. 3.—Photomicrograph (×530) showing spindle shaped cells without specific arrangement, and of low activity.

*Microscopic Pathology:* (See Fig. 3) There were a large number of spindle-shaped cells lying in a rather dense pink staining stroma. The nuclei were only moderately staining. They were rather uniform. They contained small nucleoli. A rare mitosis was seen. In some areas there was a great deal of pink-staining stroma with only a very few cells. There was no architectural arrangement. In other areas the cells were more closely packed. All of the areas sectioned showed the same picture, which was that of very low activity. These sections did not show the whorled bundles nor the bluish staining of a neurofibroma. Sections of two lymph nodes showed no tumor cells.

*Subsequent Course:* This patient has remained well eighteen months and leads a normal life, with no evidence of recurrence or metastasis. Fig. 4 shows radiograph taken eighteen months after surgery. Lung is re-expanded except in the lower third where the pleurae are thickened.

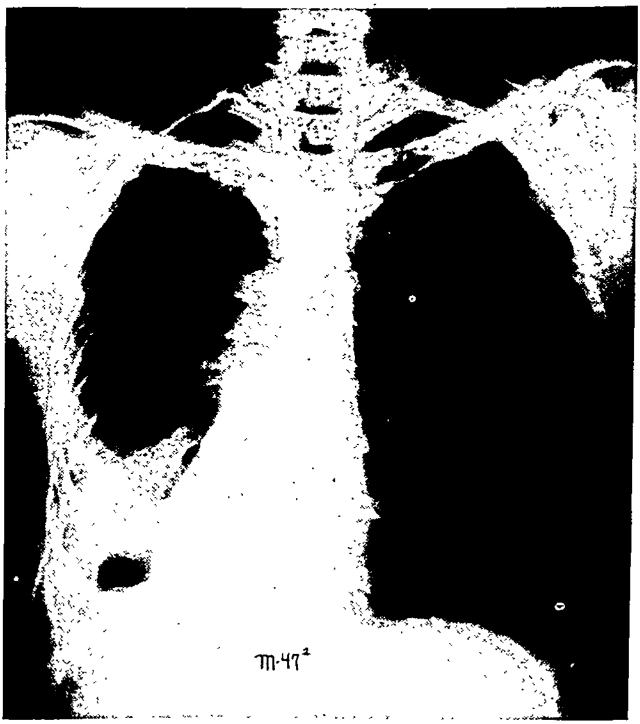

Fig. 4.—Radiograph shows left upper lobe well expanded, except in lower third where the pleurae are thickened.

## COMMENT

This tumor suggested that it originated from connective tissue of the bronchial wall, due to its physical arrangement and cellular picture. However, because of its size and extension into the lung by compression, the tumor was considered a fibroma of the lung.

## SUMMARY

1. A case of benign fibroma of lung is presented.
2. Eighteen months after removal of tumor, and the left lower lobe, there has been no recurrence.

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# Constitution and By-Laws of the American Association for Thoracic Surgery

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## CONSTITUTION OF THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

(Amended April 17, 1951)

### ARTICLE I. NAME

SECTION 1.—This Association shall be known as the American Association for Thoracic Surgery.

### ARTICLE II. OBJECT

SECTION 1.—The object of the Association shall be to encourage and stimulate investigation and study that will increase the knowledge of intrathoracic physiology, pathology, and therapy, to correlate such knowledge and disseminate it.

SECTION 2.[*]—To attain this object, the Association shall hold at least one scientific meeting every year in which free discussion shall be featured; shall conduct a Journal for the publication of the papers presented at this meeting, and other acceptable articles; and shall undertake such other activities as the Council or the Association as a whole may decide.

### ARTICLE III.[†]

### ARTICLE IV. MEMBERSHIP

SECTION 1.—There shall be four classes of members: Active, Associate, Senior, and Honorary. Admission to membership in the Association shall be by election. *There shall be no numerical limits on any class of membership. Only Active and Senior members shall have the privilege of voting or holding elective office.*[‡]

SECTION 2.—Election to all classes of membership shall *be for life, subject to the provisions of Section 3, following. (Section 3 deals with resignation and/or expulsion for cause.)*[§]

SECTION 3.—Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as a Board of Censors, may recommend the expulsion of a member on the grounds of moral or professional delinquency, and submit his name, together with the grounds of complaint, to the Association as a whole at any of the regularly convened meetings, after giving the member so accused ample opportunity to appear in his own behalf.

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[*] As amended in 1950.

[†] Amendment to delete this article passed in 1936. The article was concerned with membership in the Congress of American Physicians and Surgeons which ceased to exist.

[‡] Amendment passed, 1948.

[§] Amendment passed, first reading 1939.

## ARTICLE V. OFFICERS AND GOVERNMENT

SECTION 1.—The officers of the Association shall be a President, a Vice-President, a Secretary, a Treasurer, an Editor, and five Councilors. These ten officers and councilors shall be the governing body of the Association, and shall have full power to act on all matters, except as follows:

1. They may not alter the initiation fees or annual dues, nor levy any general assessments against the membership, except that they may, in individual cases, remit annual dues or assessments.
2. They may in no wise change the Constitution or By-Laws.
3. They may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article IV, Section 2.
4. They may not deplete the principal of the Endowment Fund.

SECTION 2.—Officers and Councilors shall be elected at the annual meeting of the Association, *and shall take office upon conclusion of the meeting.** The President and the Vice-President shall be elected for a one-year term of office and neither may be re-elected to succeed himself in the same office. The Secretary, the Treasurer, and the Editor shall be elected for a one-year term of office, and any or all may be re-elected indefinitely. The outgoing President shall automatically become a Councilor for a one-year term of office. The other four Councilors shall be elected, one each year, for a four-year term of office, but no Councilor may be re-elected to succeed himself.

SECTION 3.—Vacancies occurring among the officers and councilors during the year shall be temporarily filled by action of the Council, subject to approval of the Association at the next regularly convened meeting.

## ARTICLE VI. COMMITTEES

SECTION 1.—At the opening session of the annual meeting there shall be elected, after nomination from the floor of the Association, a Nominating Committee of three. This Committee shall prepare a slate of nominees for officers and councilors and shall present their report at the executive session of the Association.

SECTION 2.—The Council is empowered to appoint a Membership Committee, an Auditing Committee, a Program Committee, a Necrology Committee, and such other committees as may in its opinion be necessary. All such committees shall render their report at the executive session of the Association.

SECTION 3.—The Editor is empowered to appoint an Editorial Board, subject only to the approval of the Council.

SECTION 4.—The Association as a whole may authorize the Council to appoint Scientific or Research Committees for the purpose of investigating thoracic problems and may further authorize the Council to support financially such committees to a limited degree. In appointing such committees, the Council shall be governed by the provisions of the By-Laws.

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*Amendment passed, first reading 1939.

## ARTICLE VII. FINANCES

SECTION 1.—*The fiscal year of the Association shall run from the beginning of one annual meeting to the beginning of the next annual meeting.** The books of the Association shall be kept and audited on this basis.

SECTION 2.—Members shall contribute to the financial maintenance of the Association through the medium of initiation fees, annual dues, and special assessments. The amount of the annual dues and the initiation fees shall be determined by the By-Laws.

If, at the end of any fiscal year, there be a deficit in the current funds of the Association, the Council may send out notices to that effect and invite Active Members to contribute the necessary amount so that no deficit be carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment for any purpose *consistent with the objects of the Association (Article II)*, and such special assessment shall become an obligatory charge against the classes of members affected thereby.†

SECTION 3.—To meet the current expenses of the Association, there shall be available all revenue derived from annual dues, special assessments, and income from Endowment Fund, subject to the provisions of Section 4, following. Funds derived from the payment of initiation fees shall not be available for current expenses.

SECTION 4.‡—All funds derived from the payment of initiation fees shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact and to be known as the Endowment Fund. The Council is responsible for the proper management of the Endowment Fund, and may divert any surplus in the current funds of the Association into this fund, but may not withdraw any of the principal of the Endowment Fund except in accordance with the provisions of Section 6, following.

SECTION 5.‡—The income from the Endowment Fund shall be expended as the Council directs.

SECTION 6.‡—The principal of the Endowment Fund may be withdrawn, in whole or in part, under the following conditions only: The amount of principal to be withdrawn shall have been approved by the Council; it shall have been approved by a majority of the members present and voting at a regularly convened annual meeting; it shall have been tabled for one year; it shall have been finally passed by a three-fourths vote of the members present and voting at the next regularly convened annual meeting.

SECTION 7.—In the event of the dissolution of the Association, the Endowment Fund shall be distributed among national institutions of the United States and Canada in a proportion equal to the then existing ratio between the numbers of citizens of the two nations who are members of the Association.

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*Amendment passed, first reading 1939.

†As amended in 1950.

‡Final action taken in 1940.

### ARTICLE VIII. MEETINGS

SECTION 1.—The time, place, duration, and procedure of the annual meeting of the Association shall be determined by the Council, and the provisions of the By-Laws.

SECTION 2.—A special meeting of the Association may be called on one month's notice on the written request of fifteen members. The specific purposes of the meeting must be stated in the request and in the official call for the meeting.

SECTION 3.—The annual meeting of the Council shall be held at or near the close of the fiscal year.

### ARTICLE IX. AMENDMENTS

SECTION 1.—This Constitution shall in no wise be changed except by a three-fourths vote of the members present at an annual meeting, and further provided that the proposed alteration or amendment shall have been moved and seconded at a previous annual meeting, and that printed copies of the suggested alteration or amendment shall have been circulated among the members, and that the members shall have been specifically advised that such alteration or amendment will be voted upon.

## BY-LAWS

### ARTICLE I.

SECTION 1.—These By-Laws shall merely interpret the Constitution and specifically apply its principles. They shall set forth no principles not included in the Constitution.

### ARTICLE II.

SECTION 1.*—All papers read before the Association shall become the property of the Association. Authors shall leave original copies of their manuscripts with the Editor *or Reporter*, at the time of presentation, for publication in the official journal.

SECTION 2.—When the number of papers makes it desirable, the Council may require authors to present their papers in abstract, and may set a time limit on discussions.

SECTION 3.*—Members are urged to cooperate with *all* Scientific Committees of the Association.

SECTION 4.†—Attendance at Annual Meetings and participation in the scientific programs shall be optional for all Honorary and Senior Members, but it shall be expected from all Active and Associate Members.

SECTION 5.‡—While the scientific session of the annual meeting is held primarily for the benefit of the members of the Association, it may be thrown open to nonmembers who are able to submit satisfactory credentials, who register in a specified manner, and who pay such registration fee as may be determined and published by the Council from year to year. Such registered guests shall have the privilege of the floor in discussing papers if time permits.

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*As amended in 1950.

†Amended by addition of Sec. 4, 1946.

‡Amendment passed, 1948.

### ARTICLE IV.

SECTION 1.—Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active or Senior Members. Such nomination must have been in the hands of the Membership Committee for not less than four months, and the name of the candidate must have been distributed to the Association as a whole before final action may be taken on any new candidate for election to Active or Associate Membership. Provided the foregoing requirements have been met, and the candidates have been approved by the Membership Committee and by the Council, their names shall be presented to the Association at a regularly convened annual meeting for final action. A three-fourths vote of those present and voting shall be required to elect. Any candidate for membership in this Association who has failed of election for two successive years shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

SECTION 2.*—*There shall be no numerical limit on Active Membership.* The candidate, to be eligible, must be a citizen of the United States of America or of Canada, *unless in unusual cases this citizenship requirement shall have been waived by the Council. The candidate shall have achieved distinction in the thoracic field, or shall have made a meritorious contribution to knowledge pertaining to thoracic disease or its surgical treatment. Under usual circumstances, Active Members shall be elected from the Associate Members who have satisfied all requirements for Active Membership. Under unusual circumstances, Active Membership may be awarded to new candidates of outstanding distinction.*

SECTION 3.*—*There shall be no numerical limit on Associate Membership. The candidate, to be eligible, may be a citizen of any country. All candidates for Associate Membership shall be engaged actively in the practice of clinical medicine, or in teaching, or investigative work in the basic sciences underlying clinical practice, and all shall have demonstrated an active interest in thoracic disease. In the case of those who are engaged in the independent practice of thoracic surgery, it shall be required that they present evidence of sound basic training in general as well as thoracic surgery. Younger men, still in training, who have shown an interest in the thoracic field, and who are recommended by their preceptors for the excellence of their work, may be elected to Associate Membership. Clinicians of outstanding ability, whose interest is limited to a small fraction of the entire thoracic field, may be elected to Associate Membership.*

SECTION 4.—The number of Senior Members shall be unlimited. The candidate, to be eligible, must have been an Active Member of the Association for not less than ten years or, having been an active Member, have passed the age of sixty years, or become incapacitated by illness.

SECTION 5.*—*Honorary Membership shall be reserved for such distinguished persons as may be deemed worthy of this honor by the Council with concurrence of the Association.*

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*Amendment passed, 1948.

SECTION 6.*—The report of the Membership Committee shall be rendered at the annual Executive Session of the Association. Candidates shall be presented in groups in the following order: *Candidates for Honorary Membership; retirement of Active Members to Senior Membership; candidates for Active Membership; candidates for Associate Membership.*

SECTION 7.—The Council shall recommend that any member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from *three*† consecutive annual meetings, shall have his membership terminated.

## ARTICLE V.

SECTION 1.—The President of the Association shall perform all duties customarily pertaining to the office of President. He shall not only preside at all meetings of the Association, but also at all meetings of the Council. The President shall be elected from the Active Members of the Association.

SECTION 2.—The Vice-President of the Association shall perform all duties customarily pertaining to the office of the Vice-President, not only as to the Association, but also as to the Council. The Vice-President shall be elected from the Active Members of the Association.

SECTION 3.—The Secretary of the Association shall perform all duties customarily pertaining to the office of Secretary. He shall serve not only as Secretary of the Association but also as Secretary of the Council. The Secretary shall be elected from the Active Members of the Association.

SECTION 4.—The Treasurer of the Association shall perform all duties pertaining to the office of Treasurer. He shall not only serve as Treasurer of the Association but shall also serve as custodian of the Endowment Fund. The Treasurer shall be elected from the Active Members of the Association.

SECTION 5.‡—The Editor of the Association shall be the Editor of the official Journal and shall, ex officio, be the Chairman of the Editorial Board. The Editor shall be elected from the Active or Senior Members of the Association.

SECTION 6.—The Councilors of the Association shall hold office as specified in the Constitution. They may be elected from either the Active Members or the Senior Members of the Association.

SECTION 7.—In the event of a vacancy occurring in the office of President, the Council shall advance the Vice-President to the Presidency and appoint a new Vice-President under the Provisions of Article V, Section 3, of the Constitution.

## ARTICLE VI.

SECTION 1.—The Nominating Committee shall consist of three Active or Senior Members elected in accordance with the provisions of Article VI, Section 1, of the Constitution. The Council shall instruct the Committee as to the vacancies which are to be filled by election.

SECTION 2.—The Membership Committee shall consist of five Active or Senior Members appointed in accordance with the provisions of Article VI, Section 2, of the Constitution. The Council may appoint not more than one of its own members to serve on this Committee. The duties of the Membership Committee are to investigate all candidates for membership in the Association and to report their findings as expeditiously as possible to the Council through the Secretary of the Association. This Committee is also charged with searching the literature of this and other countries to the end that proper candidates may be presented to the Association for consideration. Appointment to this Committee shall be for a period of one year, and not more than three of the members may be reappointed to succeed themselves. *This Committee is also charged with maintaining a record of membership attendance and participation in the scientific programs and reporting to the affected members and to the Council any deviations from the requirement of Article II, Section 4, of these By-Laws.**

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*Amendment passed, 1948.

†Amended in 1946.

‡As amended in April, 1950.

SECTION 3.—The Auditing Committee shall consist of three Active or Senior Members appointed in accordance with the provisions of Article VI, Section 2, of the Constitution. None of these may be selected from the officers or councilors of the Association. Their duty shall be to audit the accounts of the Association at the end of each fiscal year and render their report to the Council of the Association through the Secretary. Appointments to this Committee shall be made for a one-year term. Not more than two members may be reappointed to succeed themselves.

SECTION 4.—The Program Committee shall consist of five members: The President of the Association, the Secretary of the Association, the Editor of the Association, and two members at large, one of whom shall be resident at or near the place of annual meeting. The duties of this Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

SECTION 6.—The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be, ex officio, the chairman of this board and shall be privileged to appoint and idefinitely reappoint such members of the Association, regardless of class of membership, and such nonmembers of the Association as in his opinion may be best calculated to meet the editorial requirements of the Association.

SECTION 7.—When Scientific or Research Committees are authorized by the Association, the Council shall appoint the Chairmen of these Committees, with power to organize their committees in any way best calculated to accomplish the desired object, subject only to the approval of the Council. Financial aid rendered to such Committees shall not exceed such annual or special appropriations as may be specificially voted for such purposes by the Association as a whole.

### ARTICLE VII.

SECTION 1.—Honorary Members of the Association are exempt from all initiation fees, dues, and assessments.

SECTION 2.—Annual dues for Active Members shall be $25.00.

SECTION 3.—Annual dues for Associate Members shall be $20.00.

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*Amended by addition, 1946.

SECTION 4.[*]—Senior Members are exempt from dues.

SECTION 5.—Initiation fee for those elected directly to Active Membership shall be $15.00.

SECTION 6.—Initiation fee for those elected to Associate Membership shall be $10.00. If and when an Associate Member is elected to Active Membership, he shall pay an additional $5.00 initiation fee.

SECTION 7.[†]

SECTION 8.—Income from the Endowment Fund shall be expended as the Council directs.

SECTION 9.—Associate and Active Members must subscribe to THE JOURNAL OF THORACIC SURGERY to retain their membership status.

SECTION 10.[‡]—Senior Members may retain their membership status without the payment of annual dues and subscription to THE JOURNAL OF THORACIC SURGERY is optional.

(Note.—Bills for membership dues and for subscriptions to THE JOURNAL OF THORACIC SURGERY WILL BE MAILED TO MEMBERS BY THE TREASURER AFTER THE ANNUAL MEETING.)

## ARTICLE VIII.

SECTION 1.—When the Association convenes for its annual meeting, it shall immediately go into executive session, but the business at this session shall be limited to:

1. Election of Nominating Committee.
2. Appointment of necessary committees.
3. Miscellaneous business of an urgent nature.

SECTION 2.—The annual executive session of the Association shall be held at the opening of the afternoon session of the second day of the meeting. The order of business shall be:

1. Reading of the minutes of the preceding meetings of the Association and Council.
2. Report of the Treasurer for the last fiscal year.
3. Report of the Auditing Committee.
4. Report of the Treasurer for the current year to date.
5. Report of the Necrology Committee.
6. Report of the Program Committee.
7. Action on amendments to the Constitution and By-Laws.
8. Action on recommendations emanating from the Council.
9. Unfinished Business.
10. New Business.
11. Report of the Membership Committee.
12. Election of new members.
13. Report of the Nominating Committee.
14. Election of officers.

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[*] Amendment passed in 1944.
[†] Section 7 deleted in 1950.
[‡] Amendment passed in 1944.

SECTION 3.—At the annual meeting of the Council the order of business shall be:

1. Reading of minutes.
2. Treasurer's report.
3. Appointment of committees.
4. Determination of instructions to be given to Auditing, Membership, Necrology, and other committees except Program Committee.
5. Determination of details in connection with the annual meeting.
6. Appointment and instructions to the Program Committee.
7. Unfinished Business.
8. New Business.

SECTION 4.—There shall be an annual meeting of the Council.

### ARTICLE X.

SECTION 1.—These By-Laws shall in no wise be changed except by a two-thirds vote of the members present at the annual meeting or a properly convened meeting of the Association, and further provided that the proposed action or amendment shall have been moved and seconded by not less than three of the members in a properly convened annual or special meeting of the Association.

SECTION 2.—These By-Laws may be suspended in whole or in part for a period of not more than twelve hours by a unanimous vote of those present at any regularly convened meeting of the Association.

# The Journal of Thoracic Surgery

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## Original Communications

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# MINUTE CARCINOMA OF THE MAJOR BRONCHI

## A FOLLOW-UP REPORT

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IN A paper read before the American Association for Thoracic Surgery on April 28, 1947, we cited, in abstract, two unusual case reports.[1] These concerned two patients in whom resectional therapy had been performed on the basis of a microscopic diagnosis of pulmonary carcinoma, made on examination of a specimen removed at bronchoscopy. A pneumonectomy had been performed on one patient; on the other, a middle lobe lobectomy. No carcinoma could be found in the surgical specimens, and, on postoperative bronchoscopic examination, there was no evidence of residual malignancy in the patient upon whom a lobectomy had been performed. As far as we were able to determine, these were the first such cases to be reported in the literature.

The patient upon whom a pneumonectomy had been performed has remained well for a follow-up period of six years. The subsequent history of the second patient warrants a complete case report.

### CASE REPORT

M. W. (516547) was admitted to The Mount Sinai Hospital on Feb. 12, 1944, complaining of cough of two years' duration. Cough was occasionally paroxysmal in type, especially at night. There was virtually no sputum. Roentgenographic examination, performed on a number of occasions before admission, revealed a persistent shadow in the region of the right middle lobe. Bronchoscopic examination was performed, and a specimen removed from the right middle-lobe bronchus was reported as squamous-cell carcinoma (Fig. 1). On February 23, under cyclopropane-ether-intratracheal anesthesia, a thoracotomy was performed through an anterior incision. Numerous adhesions were found between the upper, middle, and lower lobes, as well as medially on the pericardium, and anteriorly and laterally to the thoracic wall. The middle lobe was atelectatic; the upper and lower lobes were normal. No mass could be felt either in the middle lobe or at the proximal end of the middle lobe bronchus. The middle lobe was separated from the upper and lower lobes, and resected. Immediate examination of the specimen revealed no gross evidence of tumor. Therefore,

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an additional segment of the middle lobe bronchus was excised. This, too, did not contain any neoplastic tissue on gross examination nor upon subsequent microscopic examination. Because of doubt as to the advisability of performing any further resection, the bronchoscopist was summoned to examine the bronchial tree immediately. He could find no evidence of carcinoma in the bronchial tree. He removed a specimen from the middle lobe orifice, which was later reported as "Fragments of bronchial mucosa, showing chronic inflammation and a small area of cell atypism of the surface of the epithelium. No evidence of tumor."

Because of the absence of tumor in the surgical specimen and negative bronchoscopy (during operation), it was decided not to perform any additional resection. The stump of the middle-lobe bronchus was sutured, and the thorax closed with underwater drainage.

Postoperatively, an empyema developed. This was drained, following which convalescence was uneventful.

Pathologic examination of the surgical specimen was reported as follows: "The specimen consisted of a right middle lobe measuring 8.5 by 6 by 2 cm. The pleural surface showed a few fibrous adhesions near the hilum; otherwise it was smooth. The parenchyma was collapsed and bluish red in color. The bronchi were slightly widened; the mucosa was pale pinkish white and not thickened. The pulmonary vessels showed no changes. Separately received was a small piece of tissue containing segments of several bronchi. The mucosa of these was moderately reddened and slightly thickened."

Serial sections were cut from the right middle lobe bronchus for microscopic examination. No evidence of malignancy could be found. The final diagnosis was "right middle lobe, showing atelectasis and peribronchial and perivascular fibrosis." The original bronchoscopic specimen, upon which the diagnosis of squamous-cell carcinoma had been made, was reviewed by a number of pathologists, all of whom concurred in the diagnosis of carcinoma (Fig. 1).

On April 3, a phrenicectomy was performed to aid in obliteration of the empyema space, and on April 10, another bronchoscopic examination was performed. There was thick secretion in the right main and lower lobe bronchi. There was no recurrent flow. The right middle-lobe orifice showed a healed defect on its lower tip. The stump of the right middle-lobe bronchus had smooth walls and was closed at its lower end. There was some thickening of the lateral wall of the middle-lobe bronchus. A specimen removed from this area revealed "fragments of bronchial mucosa, showing metaplastic squamous-cell epithelium and a few isolated irregular cells. No evidence of tumor."

The patient was discharged on April 12, 1944, and remained under observation for several years. His cough persisted, but was no worse than it had been prior to operation. Roentgenographic examination of the chest showed no changes.

On Feb. 10, 1949, he was admitted to the Presbyterian Hospital, complaining of cough, anorexia, and a weight loss of 30 pounds. He stated that this cough had begun in 1947 and had become progressively worse. At times the cough was paroxysmal and associated with vomiting of blood. He also stated that he had had a pleurisy eighteen months before admission.

Physical examination revealed the signs of chronic illness. There were dullness and diminished breath sounds over the right lower lobe. The hemogram was within normal limits; the sputum was negative for tubercle bacilli on three examinations. Roentgenographic examination of the chest showed a 4 by 3 by 4 cm. mass at the right hilum.

Bronchoscopy performed on February 21 was reported as follows: "Just below the carina there was marked thickening of the mucous membrane and superficial ulceration of the right main bronchus. The thickening caused some narrowing of the bronchus. The lesion did not appear to be a tumor in that there was no fixation or rigidity." A specimen removed for biopsy revealed carcinoma. Exploratory thoracotomy was advised, but the patient left the hospital on March 4.

He did not return until July 19, when he was readmitted. At that time his general condition was unchanged. Bronchoscopic examination revealed that the carcinoma was thickened, irregular, and ulcerated. The same process was present in the trachea, opposite to, and above, the carina, extending from the previously described lesion in the right main bronchus.

The carcinoma now was inoperable, and radiation therapy was administered from July 23 to August 17. In December, 1949, roentgenographic examination showed an increase in the size of the mass at the right hilum.

He was again admitted to the Presbyterian Hospital on Feb. 16, 1950, with metastases to the cervical and axillary lymph nodes and questionable metastases to the cervical vertebrae. The alkaline phosphatase was 8.8. He was discharged on March 25, and testosterone was prescribed.

This patient was next seen at Montefiore Hospital, where he was admitted on Sept. 17, 1950, in a preterminal condition. He rapidly deteriorated and died on September 27.

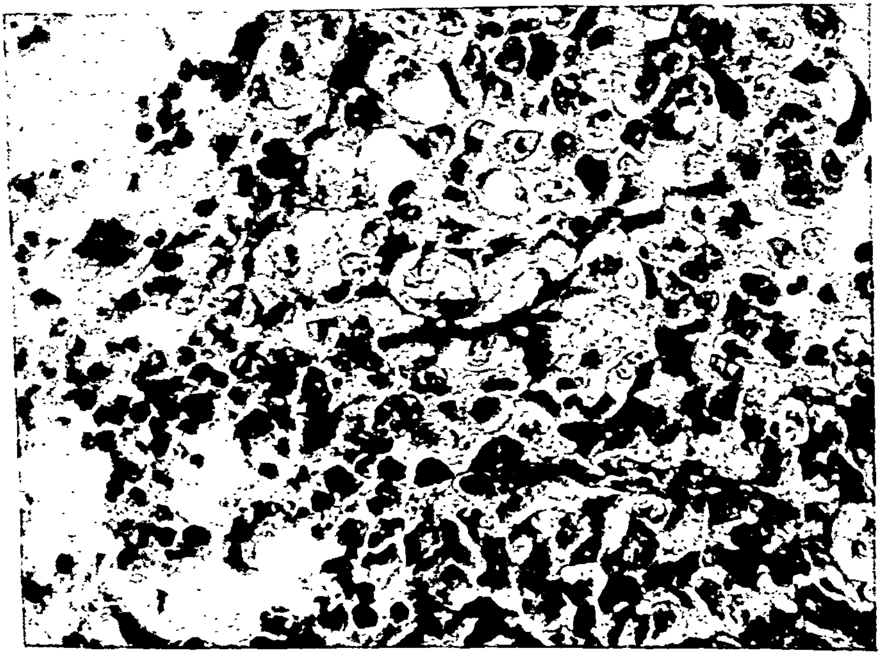

Fig. 1.—Photomicrograph of the specimen removed at the first bronchoscopy upon which the diagnosis of carcinoma was made.

Post-mortem examination revealed the following: anaplastic bronchogenic carcinoma of the right lung with extension to the trachea; metastases to heart, pancreas, liver, adrenals, kidneys, skeleton, lymph nodes of peribronchial, left axillary, and periaortic chains; focal pneumonia and focal fibrosis of the lungs. The lung was described as follows: "The distal one inch of the trachea presented a thickened granular mucosa which was markedly firm in consistency and slightly congested in areas. This granulation and thickening extended down into the right main-stem bronchus. The bronchi of the right lung could not be traced further than this point except for several in the lower lobe. The right middle-lobe bronchus was healed by a scar. A tumor mass occupied the lung around the hilum."

The occurrence of "minute" carcinoma of the lung has been described by Petersen, Hunter, and Sneeden.[2] Their specimens were in the parenchyma of the lung, but they believed that they arose from bronchial elements. "Minute" carcinoma of the main bronchi have not been described, but it is evident that they may be present at some time in the development of a carcinoma.

Papanicolaou and Kropowska[3] have reported a carcinoma in situ of the right lower bronchus. Although four specimens of sputum and six bronchial washings were positive for carcinoma cells, bronchoscopy was negative. The patient died from emphysema, anoxemia, and lobar pneumonia. Gross examination of the lungs revealed no evidence of malignancy. On careful examination with many serial sections of the bronchial tree, a minute epidermoid carcinoma in situ was found in the bronchus of the right lower lobe.

Despite the *definite bronchoscopic diagnosis* of malignancy made in both of our cases, we originally reported them with some hesitancy because of the inability to substantiate the diagnosis by the finding of the carcinoma in the resected specimens. In the patient upon whom lobectomy was performed, there was the possibility that the actual site of the tumor had not been removed. Postoperative bronchoscopic examination, with removal of a specimen from the stump of the middle-lobe bronchus, still failed to reveal any evidence of malignancy. The subsequent course of this patient, with development of a carcinoma a number of years later, apparently proves the correctness of the original diagnosis. The tumor was undoubtedly one of slow growth and, as is so often seen in pulmonary malignancy, did not cause symptoms until the later stages. It is most likely that if a pneumonectomy had been performed instead of a lobectomy the patient would have been cured of a pulmonary carcinoma, as was the other patient, who has remained well for six years.

The thoracic surgeon must rely completely upon the pathologist's diagnosis made on the microscopic examination of specimens removed during bronchoscopy. It has been our experience that when, prior to surgery, a *definite* diagnosis of malignancy is made on examination of a specimen removed during bronchoscopy, it has invariably been substantiated (except in the cases cited) by later examination of the surgical specimen. On occasions, the pathologist is not able to make a definite diagnosis, and then additional bronchoscopy or exploratory thoracotomy is indicated. In both of our patients, the diagnosis of malignancy was a definite one and concurred in by every pathologist who examined the slides.

## SUMMARY AND CONCLUSIONS

A follow-up report of two cases previously cited is presented.

Bronchoscopic specimens in both cases were reported as carcinoma. One patient had a pneumonectomy, and has remained well for six years (no carcinoma could be found in the resected lung). At operation on the second patient, no gross tumor was found, and bronchoscopy during the operation revealed no evidence of disease. Therefore only a lobectomy was performed. Subsequent bronchoscopy was also negative. Five years later, clinical symptoms appeared, and bronchoscopy revealed a carcinoma of the main bronchus of the lung previously operated upon. The patient died six and one-half years after the original diagnosis had been made.

If the pathologist makes an unequivocal diagnosis of malignancy upon examination of a specimen removed from a major bronchus, the surgeon should perform a pneumonectomy even though no gross evidence of malignancy is found at operation.

We wish to thank Dr. William Stearns for permission to use the records of the Presbyterian Hospital, and Dr. Harry M. Zimmerman for the use of the post-mortem records of Montefiore Hospital.

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1. Neuhof, H., and Aufses, A. H.: Cancer of the Lung, J. THORACIC SURG. **17**: 297, 1948.
2. Petersen, A. B., Hunter, W. C., and Sneeden, V. D.: Histological Study of Five Minute Pulmonary Neoplasms Believed to Represent Early Bronchogenic Carcinoma, Cancer **2**: 991, 1949.
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# MINUTE PERIPHERAL PULMONARY TUMORS

## A STUDY OF EIGHT CASES

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THE discovery of clinically unsuspected pulmonary neoplastic disease in routine autopsy and surgical material is a very infrequent occurrence. This may be partially explained by the fact that the parenchymatous tissue of the lung does not lend itself readily to visual examination, with the result that the pathologist is forced to depend largely on palpation for the identification of diseased areas. Furthermore, despite the large volume of the organ, relatively few blocks often taken in haphazard fashion are usually removed for microscopic study. These limitations associated with the gross examination do not wholly account for the discovery of the tumors to be described below, since they all represented accidental microscopic findings and were undoubtedly too minute in size to be perceived with the naked eye.

We have collected eight of these pulmonary neoplasms, all identical in their microscopic appearance, and all representing incidental findings in our autopsy and surgical specimens. It is felt that the reporting of these cases will be of value, not only because of the small number of recorded instances of similar atypical cellular proliferation (Table I), but chiefly because our investigations have convinced us of their relatively benign character. This latter view finds support in a study by Womack and Graham,[1] but is in complete disagreement with the majority of those who have described similar epithelial overgrowths.[2-6]

### MATERIAL

Six of our eight cases were incidental observations at autopsy of persons dying from a variety of causes, while the remaining two were from surgical specimens removed because of long-standing severe bronchiectasis (Table II). All tissue was fixed in 10 per cent formalin, and in addition to the usual hematoxylin and eosin stain, the sections containing tumor were stained with the following methods: Weigert van Gieson, Wilder's reticulum, McManus' periodic acid Schiff, and Lison's argentaffine techniques. Several of the blocks were serially sectioned in an attempt to trace the origin of the tumor and to gain some idea of its relative size. These sections were cut at a thickness of 6 microns, and as many as 225 sections were obtained from a single block.

### REPORT OF CASES

CASE 1.—L. A. (A44-242), an 81-year-old woman, who was a known cardiac with a history of bronchiectasis of several years' duration, expired from bronchopneumonia after a four-day illness. The chief findings at autopsy were bilateral bronchopneumonia, bronchiectasis, fibrosis and emphysema.

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From the State University of New York, Medical Center at Syracuse University.
Contribution of the authors from the Department of Pathology, State University of New York, Medical Center at Syracuse University.
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Table I. Previously Recorded Minute Pulmonary Neoplasms

| DATE | AUTHOR | AGE (YR.) | SEX | CAUSE OF DEATH | GROSS PULMONARY DISEASE | LOCATION OF TUMOR |
|---|---|---|---|---|---|---|
| 1921 | Gray and Cordonnier | 78 | F | Fracture of femur | Calcified apical nodule<br>Bronchiectasis? | |
| 1930 | Karsner and Saphir | | | | Bronchiectasis | |
| 1941 | Womack and Graham | 34 | M | | Congenital cystic disease | Left lung |
| 1941 | Womack and Graham | 48 | M | Congenital cystic disease | | Left lung |
| 1941 | Womack and Graham | 39 | M | Congenital cystic disease | | Right lower lobe |
| 1943 | Stewart and Allison | 32 | F | Alive and well | Bronchiectasis | Left lower lobe |
| 1949 | Petersen et al. | 69 | F | Carcinoma of cecum | Bronchopneumonia<br>Purulent bronchitis<br>Bronchiectasis | Left upper lobe |
| 1949 | Petersen et al. | 54 | M | Hypertensive cardiovascular disease | Bronchopneumonia | |
| 1949 | Petersen et al. | 74 | F | Bilateral pyonephrosis | Acute bronchitis<br>Atelectasis | |
| 1949 | Petersen et al. | 52 | F | Generalized peritonitis | Bronchopneumonia<br>Atelectasis<br>Bronchiectasis | |
| 1949 | Petersen et al. | 70 | F | Bronchopneumonia<br>Purulent bronchiectasis | Bronchopneumonia<br>Purulent bronchiectasis | |
| 1951 | Spain and Parsonnet | 95 | F | Encephalomalacia | Bilateral subapical fibrosis<br>Diffuse emphysema<br>Enlarged hilar nodes | Both lower lobes<br>Right upper lobe<br>Hilar node |

Table II. Authors' Series Minute Pulmonary Neoplasms

| CASE NUMBER | AGE | SEX | CAUSE OF DEATH | GROSS PULMONARY DISEASE | LOCATION OF TUMOR |
|---|---|---|---|---|---|
| 1 | 81 | F | Bronchopneumonia | Bronchopneumonia<br>Bronchiectasis<br>Fibrosis<br>Emphysema | |
| 2 | 48 | F | Rheumatic heart disease | Bronchopneumonia | |
| 3 | 47 | F | Acute hemorrhagic pancreatitis | Lower lobe atelectasis | One lower lobe |
| 4 | 64 | F | Carcinoma of the cervix | Fibrosis, left apex<br>Emphysema | Left upper lobe |
| 5 | 68 | F | Cerebral thrombosis | Bronchiectasis<br>Thickened visceral pleura | |
| 6 | 43 | M | Alive and well | Bronchiectasis | Right lower lobe |
| 7 | 30 | F | Alive and well | Bronchiectasis | Left lingual lobe |
| 8 | 70+ | F | Heart failure<br>Arteriosclerotic heart disease | Bronchopneumonia | Left lower lobe |

Case 2.—M. S. (A48-125), a 48-year-old woman with a four-year history of cardiac failure on a rheumatic basis, died following an embolism to the left lower extremity. At autopsy the principal findings were severe mitral stenosis, chronic pericarditis, recent splenic infarction and an arterial embolus in the left lower extremity. Examination of the lungs revealed bilateral bronchopneumonia.

Case 3.—H. C. (A48-297), a 47-year-old woman with a history of gall bladder disease, was admitted to the hospital with a history of abdominal pain of four days' duration. Exploratory laparotomy disclosed an acute hemorrhagic pancreatitis, and the patient expired a few hours after the surgery. Significant autopsy findings were a generalized peritonitis with fat necrosis, acute hemorrhagic pancreatitis, and an early lower nephron nephrosis. *Slight bilateral lower lobe atelectasis was noted in the lungs.*

CASE 4.—H. V. (A49-43), a 64-year-old woman, died as a result of advanced carcinoma of the cervix of two years' duration. At autopsy the chief findings were carcinoma of the cervix with extension to the bladder, rectum, ureters, and pelvic nodes. An acute bilateral pyelonephritis, and a terminal endocarditis of the mitral valve were present. Gross *examination of the lungs revealed some emphysema and fibrosis in the left apical region.*

CASE 5.—C. K. (C. I. H. A51-8), a 68-year-old woman, was admitted to the hospital for nursing care following a cerebrovascular accident five weeks previously. During her hospital stay she became incontinent, developed cystitis, pyelonephritis, and died in uremia. In addition to the urinary pathology, a large focus of encephalomalacia was noted at autopsy *in the left cerebral hemisphere.* Some *bronchiectasis and thickening of the right* visceral pleura were also described.

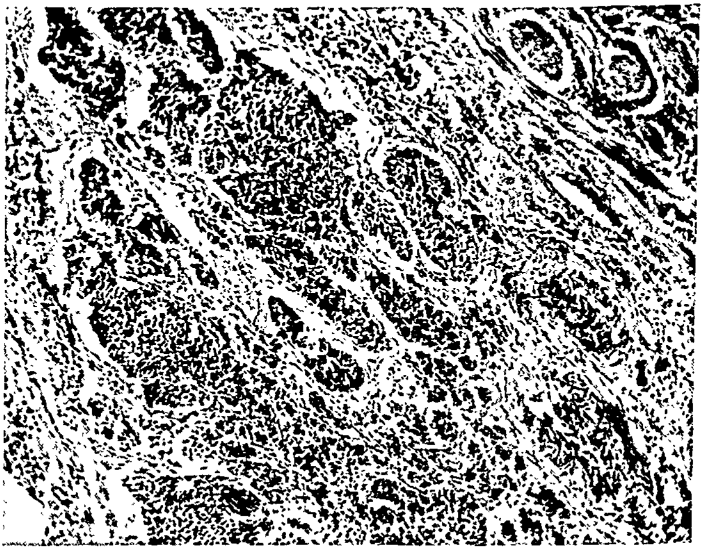

Fig. 1.—Case 7. Low-power view of the largest observed single area of tumor. Note the tendency of the cell nests to lie in spaces. Hypertrophied muscle bundles can be seen in the upper right corner. (×112.)

CASE 6.—C. S. (50-1880), a 43-year-old man, underwent a right lower lobectomy for severe bronchiectasis of seven years' duration. Gross examination of the resected lobe revealed a fusiform dilatation of the larger radicles of the bronchial system while the smaller branches were distended to form blind-end cysts. The patient had an uneventful convalescence and is alive and well at the present time (one year and a half after surgery).

CASE 7.—M. P. (50-2489), a 30-year-old woman, underwent a left pneumonectomy for bronchiectasis of two years' duration. Grossly the lower lobe and lingual bronchi and bronchioles were characterized by diffuse fusiform dilatations. A solitary 1 cm. calcified focus was noted at the apical portion of the lower lobe. The patient had an uneventful recovery and is alive and well at present (one year after surgery).

CASE 8.—M. S. (A51-81), a 70-year-old woman suffering from chronic congestive heart failure on an arteriosclerotic basis, was admitted to the hospital with a diagnosis of pulmonary infarction. The patient expired a few minutes following arrival, and at autopsy the significant findings were myocardial hypertrophy and fibrosis with severe sclerosis of the coronary arteries. Gross examination of the lungs disclosed bilateral lobular pneumonia and slight pulmonary edema. Bilateral chronic pleuritis was described.

## PATHOLOGIC FEATURES

The tumor foci were readily apparent under low-power magnification, and were peripherally and usually subpleurally located. As will be noted in Table II, it was possible to further localize the tumor in only four cases, but it would appear that either lung and any lobe may be affected. The characteristic and constant microscopic pattern of these tumors consisted of sharply circumscribed epithelial nests enmeshed in a dense fibromuscular stroma (Fig. 1). These

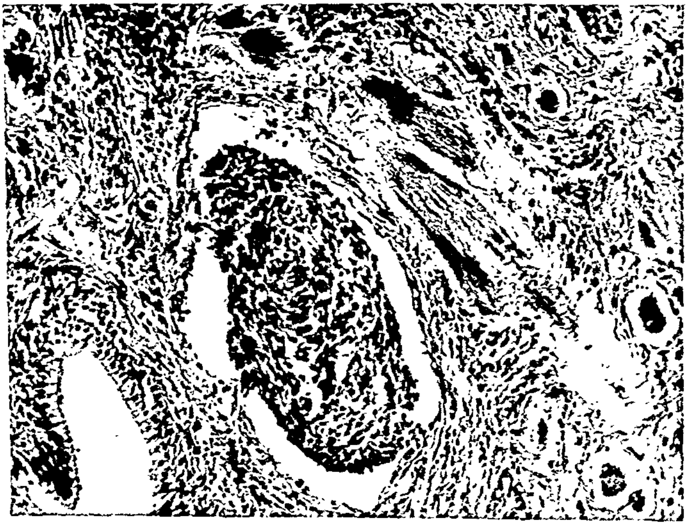

Fig. 2.—Case 7. Nest of tumor cells within a clear space showing points of penetration of the capillary stalks into the tumor. The adjacent muscle bundles are particularly prominent. (×170.)

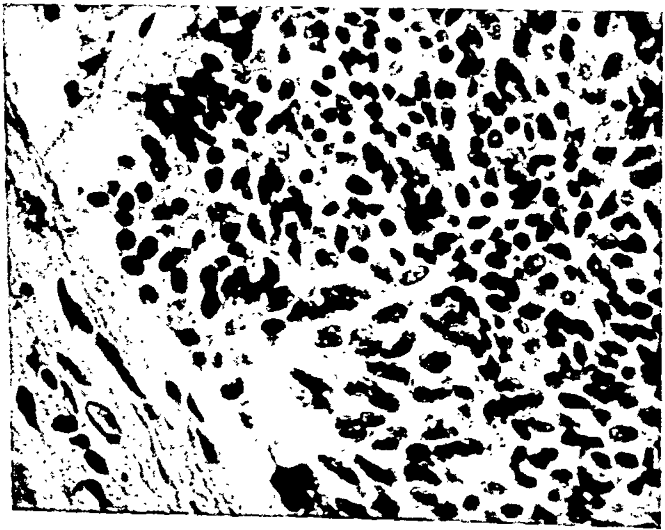

Fig. 3.—Case 7. High-power view of the individual cells comprising the tumor. A mixture of round and spindle-shaped forms are noted but mitotic figures cannot be identified. (×650.)

tumor nests varied somewhat in size, with 100 microns in diameter being an average size, and 250 microns the upper limit. A clear space separated the tumor nests from their surrounding stroma (Fig. 2), and most observers have regarded these spaces as lymphatic channels—a fact largely responsible for the impression that these neoplasms are malignant in nature. The individual cells composing the nests were characterized by prominent vesicular nuclei which were both spindle and round in shape (Fig. 3). Nucleoli were not discernible, and at no time were mitoses noted in any of the cells, including those tumors which were serially studied. The cytoplasm of the tumor cells was sparse, faintly acidophilic, and indefinite in outline. Blood capillaries and a small amount of collagen and reticulin fibers could be identified within many of the nests with the use of appropriate stains. The point of attachment of these connective tissue stalks to the marginal stroma was sometimes noted (Fig. 2). It is our belief that these pseudolymphatic spaces are artificial in nature produced by shrinkage during the processes of fixation and embedding. This conclusion is supported not only by the demonstration of the vascular stalks and cores, but also by the fact that we frequently noted a definite "sun-ray" appearance at the nests' peripheries due to loss of cohesion between the outer cells and the adjacent connective tissue (Fig. 4). A second histologic pattern, observed in four of our cases, was an intrabronchiolar polypoid type of growth (Fig. 5). The cells comprising these masses were subepithelial in location, and cytologically identical with those described above within the fibromuscular stroma. The bronchiolar epithelium was continuous over these masses, but tended to undergo a transition to a flattened spindle-shaped type of cell (Fig. 6). In the study of serial sections we failed to find unquestionable evidence that these intrabron-

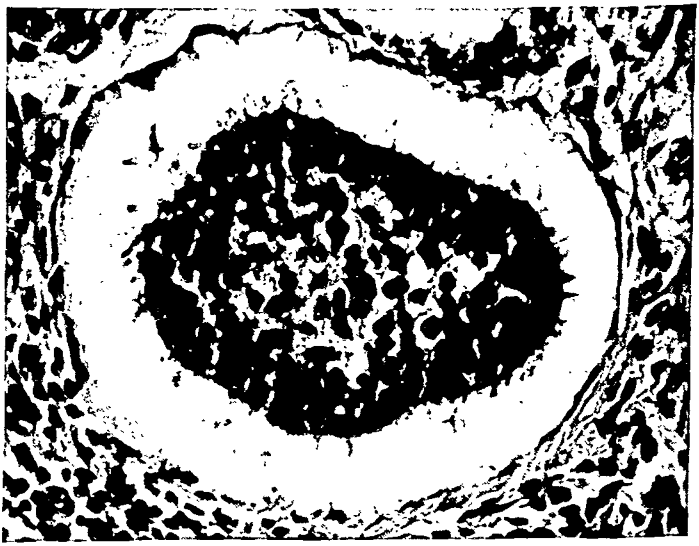

Fig. 4.—Case 6. "Sun-ray" appearance produced by shrinkage of the tumor nest away from its adjacent connective tissue. (×500.)

chiolar lesions arose from the covering epithelium, i.e., at no point was the covering epithelium composed of these tumor cells. Direct cellular continuity between these masses and the near-by stromal tumor nests could not be demonstrated.

In a consideration of the coexisting pulmonary pathology the cases will be divided into two general groups: those with bronchiectasis and those without

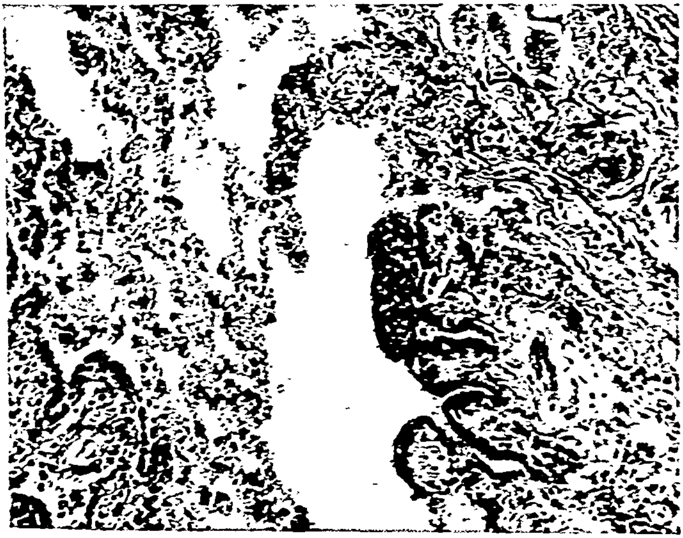

Fig. 5.—Case 5. Intrabronchiolar polypoid type of growth. This pattern was noted in four cases. (×200.)

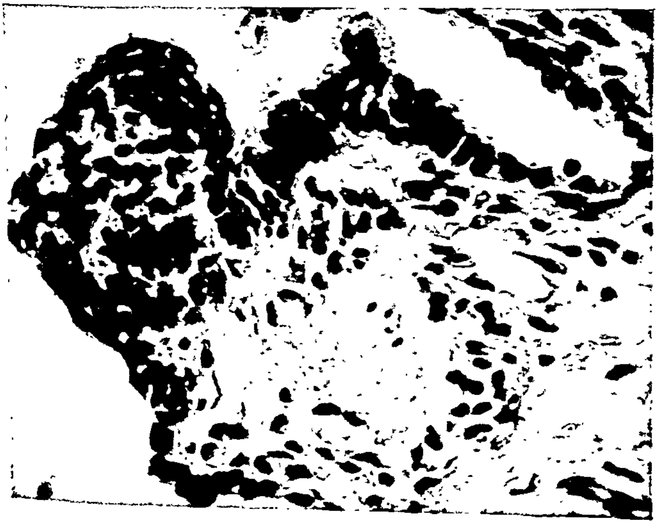

Fig. 6.—Case 7. Higher magnification of a polypoid mass within a terminal bronchiole. The transition from the normal low cuboidal epithelium to the spindle type of cell is striking. (×450.)

it. The four bronchiectatic cases in the present series had the usual dilation of the bronchial system with acute and chronic inflammation in and about their walls. The bronchial lumens contained inspissated mucus, and the mucous membrane was intact and only rarely showed evidence of squamous metaplasia. The smooth muscle coat, although extensively infiltrated with lymphocytes and eosinophiles, was well preserved. The near-by bronchioles, some of which contained tumor and some with tumor immediately adjacent, were incorporated in a sclerosing fibrous tissue growth in which hypertrophied muscle bundles were frequently prominent (Fig. 2). This latter observation suggests that, in some of the cases at least, the tumor nests may be located at the sites of obliterated bronchioles. Where alveoli could still be recognized, marked atelectasis and occasional emphysematous foci were present. Chronic inflammatory cells were not always observed in these scarred areas. Tumor was noted in the fibrous tissue surrounding an intense deposition of anthracotic pigment in one case. The medium-sized arteries were the site of rather marked intimal fibrous tissue proliferation. It was possible to localize the tumors in only two of the four cases. The tumor foci were located in the right lower lobe in Case 6, and in the left lingual and lower lobes of Case 7.

The four cases showing no gross or microscopic evidence of bronchiectasis will be individually considered. Case 2, a cardiac patient, disclosed a bronchopneumonia of recent origin at autopsy. This case is of interest in that the one section in which tumor was recognized was a subpleural atelectatic focus at some distance from the pneumonic process. This solitary neoplastic area was surrounded by fibrous tissue and scattered areas of emphysema were observed. Obliterative endarteritis was also noted throughout the atelectatic zone. Case 3, dying as a result of acute hemorrhagic pancreatitis, showed some slight, apparently agonal lower lobe atelectasis at autopsy. This case is unusual because the lone nest of tumor cells was located in a very small perivascular scar in a lung essentially free from evidence of other disease. Furthermore, this solitary tumor focus was situated at a considerable distance from either bronchi or bronchioles and was immediately adjacent to the thickened pleural surface. Case 4, who died from the complications of carcinoma of the cervix, disclosed a small fibrotic area with some calcification at the apex of the left lung. Directly beneath this probable focus of tuberculosis, multiple tumor nests in a dense connective tissue stroma, and occasional intrabronchiolar masses were noted. Some atelectasis, emphysema, and deposition of anthracotic pigment was located near by. Case 8, dying in congestive heart failure, showed bilateral bronchopneumonia at autopsy. The one section in which tumor was accidentally discovered showed several intrabronchiolar polypoid lesions and one fibrotic zone with multiple discrete tumor acini. This area was not adjacent to the pneumonic process, and its relation to the pleura could not be determined.

Although it was not possible by means of the serial section technique to trace any direct continuity between the intrabronchiolar tumor and that noted within the scarred foci, this method of study did afford some idea of the relative size of these tumors. Blocks were selected which contained the largest areas of tumor, and were sectioned according to the method described above.

Since the neoplastic foci when so studied were consistently over 0.1 cm. in length from the site where the sectioning was begun, it is reasonable to assume that there was at least a similar amount of tumor proximal to the point where the growth was originally identified. The results of this investigation indicate that the tumor may measure somewhat over 0.2 cm. in at least one plane.

## DISCUSSION

In discussing the etiologic agents which may be concerned with the production of primary lung carcinoma, Weller[7] mentioned the possible role of chronic inflammatory conditions which are accompanied by metaplasia and excessive regeneration. He stated that such conditions as bronchiectasis and chronic fibroid pneumonia might be predisposing factors, but that influenza, which some considered a causal agent, was of significance only by virtue of the chronic inflammation associated with its sequence. Wolbach and Frothingham,[8] in their report of the fatal cases in the influenzal epidemic at Camp Devens in 1918, noted evidence of repair of the bronchitis and peribronchitis as early as twelve or thirteen days after the onset of the disease. The small bronchioles had a fresh epithelium covering the underlying new connective tissue and the bronchioles appeared permanently enlarged. The authors' photomicrographs showing excessive regeneration of the epithelium in atelectatic alveoli which were undergoing repair in no way resemble the process noted in our eight cases. Pulmonary epithelial proliferation has been experimentally produced by Winternitz and associates[9] using intrabronchial insufflation of dilute hydrochloric acid. Overproduction of the epithelium occurred with the formation of polypi within the bronchioles, and extension of the epithelium into the surrounding tissue was described. Regenerating cells were identified by their size, cytoplasmic homogeneity, variation in nuclear density, and by the frequency of mitotic figures. The photomicrographs of these pathologic processes show no similarity to the changes we have described and the pictures of "extra bronchiolar invasion" are not convincing. In this connection it may be pertinent to mention an unusual case of chronic pneumonitis in an elderly woman which we recently encountered in our autopsy material. Microscopically, the pulmonary tissue showed extensive squamous metaplasia within the bronchioles and multicentric areas of invasion of surrounding alveolar spaces with epithelial atypism. Whether this case represents true metaplasia or early multicentric squamous carcinoma is not entirely clear, but it fails to show any histologic resemblance to the twenty cases of minute pulmonary neoplasm herein reviewed.

The frequency with which bronchiectasis has been noted as a concomitant pathologic finding is of considerable interest. This condition was present in four of our cases, in three of the five instances reported by Petersen and co-workers,[5] and in the single cases of Stewart and Allison,[4] and of Karsner and Saphir.[3] Furthermore, Womack and Graham[1] admit that the series of nine cases of cystic disease of the lung in which three "epithelial overgrowths" were present may be considered by some actually to be bronchiectasis. They classified their cases as congenital cystic disease because they felt there was evidence of developmental malformation of the lungs. These three cases would raise the incidence of bronchiectasis to fourteen of a total of twenty recorded cases. Because of this association, we reviewed a large number of our autopsy and surgical specimens in an attempt to find additional examples of this unusual tumor, but none were found. Furthermore, Watts and McDonald,[10] in a thorough study of the pulmonary alveolar lining of fifty human lungs removed for bronchiectasis, did not encounter epithelial proliferation of the variety described herein. Although the relationship of bronchiectasis to these tumors is at present obscure, if this condition is a factor in the pathogenesis of these tumors, certainly one should expect a rising incidence of this neoplasm in view of the increasing number of cases coming to surgery.

The most important single consideration relative to these tumors is whether or not they represent a malignant growth. The pathologist's interpretation may afford a very serious prognosis in individuals of a young age group as in Cases 6 and 7 of the present series, both being originally reported as "incidental" oat-cell carcinomas. As we have stated above, our studies have convinced us of the relative benignancy of this neoplasm—a viewpoint not in accord with most other observers. To evaluate these tumors in relation to bronchogenic carcinoma a series of thirty-nine autopsied cases of bronchogenic carcinoma were studied as a small control group. It was felt that were these minute lesions incipient bronchogenic carcinomas, they should follow the same trends as to age and sex distribution, location, microscopic appearance, and metastatic tendencies. The following is a brief summary of this survey.

### AGE

The average age of 57 years for the minute tumor group corresponded exactly with that of the bronchogenic carcinoma group. The occurrence of 20 per cent of the cases of the minute lesions below the age of 40 years is perhaps a little unusual for bronchogenic carcinoma of the usual type, but one cannot deny that incipient carcinoma might be present for some years before clinically recognized tumor results.

### SEX

Fourteen of the nineteen (70 per cent) minute tumors where the sex data were available were females. This figure contrasts sharply with a 13 per cent female incidence in the bronchogenic carcinoma series. This finding casts grave doubts on the assumption that these tumors are bronchogenic carcinoma even in the early stage.

### LOCATION

The minute lesions were consistently found in the extreme periphery of the lungs, and were apparently related to bronchioles. In contrast, the bronchogenic carcinomas, with the exception of adenocarcinomas which are frequently peripheral, were primarily related to large bronchi and were situated at or near the hilar region of the lungs.

### MICROSCOPIC FEATURES

The minute tumors in our eight cases were characterized by relative regularity of the nuclei and lack of mitotic figures. Definite origin from bronchiolar epithelium could not be demonstrated. The microscopic features of manifest bronchogenic carcinoma must be separated according to cell types. Squamous carcinoma need not be considered since there is no histologic resemblance between these two lesions. Adenocarcinomas may likewise be discarded on this basis although their peripheral location suggests some similarity. Histologically, the so-called oat-cell, undifferentiated, or spindle-cell carcinoma of the lung most closely resembles these minute tumors. Study of the oat-cell carcinomas reveals a larger cell characterized by frequent mitoses, hyperchromatic nuclei with clumping of the chromatin, and nuclear necrobiotic and pyknotic changes (Fig. 7).

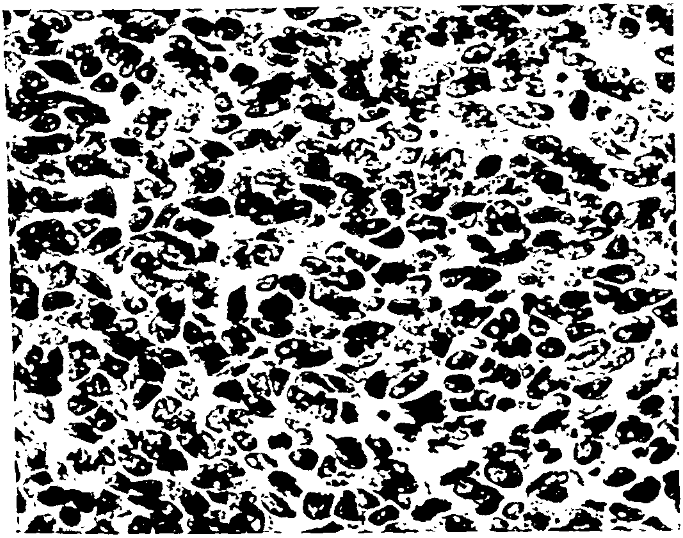

Fig. 7.—Oat-cell carcinoma for comparison with Fig. 3. The large cells show considerable pleomorphism, mitotic activity, and necrotic foci. (×650.)

## METASTASES

Metastatic tumor was found in one of the twenty cases of these minute tumors. Since in only two of our eight cases were the lymph nodes studied, these negative findings do little to clarify the ability of these minute tumors to metastasize.

From the onset of this study we have been impressed by the striking resemblance of these minute neoplasms to the carcinoid type of intestinal tumor (Fig. 8). This analogy becomes increasingly attractive by reason of the fact that the carcinoid pattern is not only the most frequently encountered variety of bronchial adenoma, but also is considered to manifest the lowest degree of invasiveness.[11] Common features to both the carcinoid tumors and the minute neoplasms herein reported are sharply circumscribed nests of uniform epithelial cells lacking evidence of mitoses, nuclear hyperchromatism, and necrobiosis. In addition the pseudolymphatic spaces and capillary stalks within the cell nests are frequently noted in carcinoid tumors elsewhere. Certain clinical characteristics of these minute pulmonary tumors such as their overwhelming preponderance of women (fourteen of the nineteen cases where these data are available) are much more suggestive of bronchial adenoma than bronchogenic carcinoma. The presence of several nests of tumor cells in a hilar node of one case does not invalidate the analogy since the extremely low grade of malignancy of the bronchial adenoma is well recognized. The argyrophile reaction was used on the two largest of these minute tumors with negative results, but it must be pointed out that true bronchial adenomas of the carcinoid type are rarely argyrophilic.[12] While we cannot state that these small peripheral tumors are identical with the large bronchial adenomas, we feel that their similarity in histologic pattern and in behavior to the carcinoid variety of adenoma is too great to be ignored (Fig. 9).

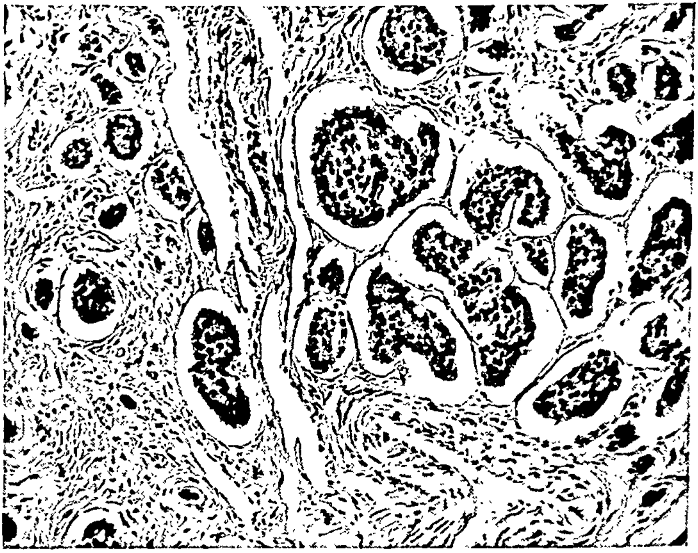

**Fig. 8.—Intestinal carcinoid demonstrating the tendency of the cell nests to occur within spaces similar to the tumors herein reported. (×165.)**

Since both the medical literature and the material in our files fail to indicate that one finds large tumors in the peripheral pulmonary fields corresponding in incidence, sex distribution, location, and cytologic structure to these minute tumors, one must assume that these relatively benign tumors remain small over a long period of time. Follow-up studies are largely precluded because the majority of the twenty reported cases have been incidental autopsy findings. Stewart and Allison[4] described a focus of tumor, identical in structure with those in our series, in a 32-year-old woman who underwent a lobectomy for bronchiectasis of long duration. The tumor which was accidentally discovered upon microscopic examination of the surgical specimen measured 0.1 cm. by 0.04 cm., and was located 0.5 cm. from the lateral surface of the lung. The authors considered this tumor to be an oat-cell carcinoma but were impressed by its close resemblance in many ways to basal cell carcinoma as seen in the skin. In a recent communication Stewart[13] has informed us that this patient is well at present and free from any evidence of metastasis, now over nine years since the date of surgery. An attempt is being made at present to secure follow-up data on the three cases occurring in young individuals reported by Womack and Graham.[1] Cases 6 and 7 of our series who underwent pneumonectomy and lobectomy, respectively, for bronchiectasis, are now alive and well although it is slightly over one year in each instance since the time of surgery.

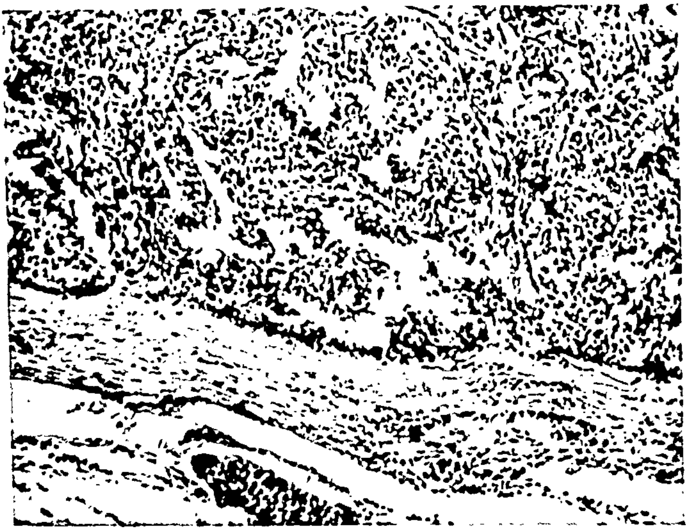

Fig. 9.—Large bronchial adenoma of the carcinoid type showing local invasiveness and a cellular pattern similar to the minute neoplasms herein described. (×170.)

## SUMMARY

Eight new cases of an unusual type of minute pulmonary neoplasm have been analyzed in respect to their clinical and pathological features. Evidence is presented to show that these tumors are relatively benign in nature, resembling most closely the pattern seen in the carcinoid type of bronchial adenoma. In addition to their histologic structure, other common features of these tumors were their minute size, accidental microscopic discovery, subpleural location, and overwhelming predilection for females. The concomitant occurrence of bronchiectasis in the majority of the recorded cases suggests that this condition may be a factor in the pathogenesis of this atypical type of epithelial cell proliferation.

The authors wish to express their appreciation to Dr. Walter F. Bugden for permission to report Cases 6 and 7 of this series, and also to Mr. Louis Giorgianna of the Department of Photography for the illustrative material herein reproduced.

## REFERENCES

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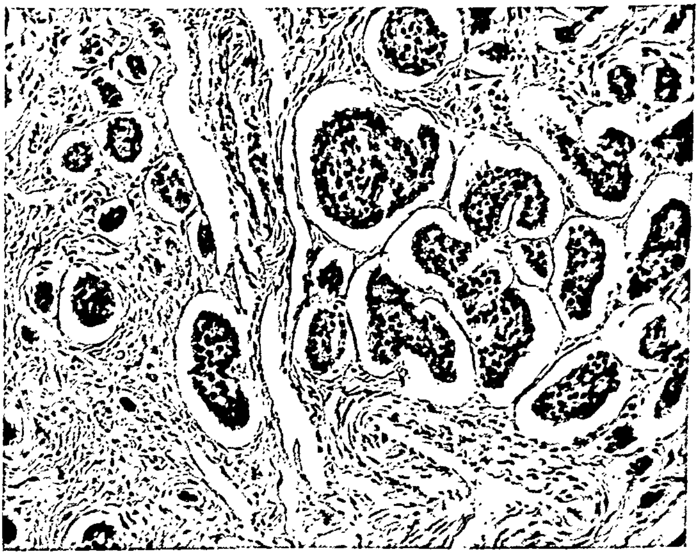

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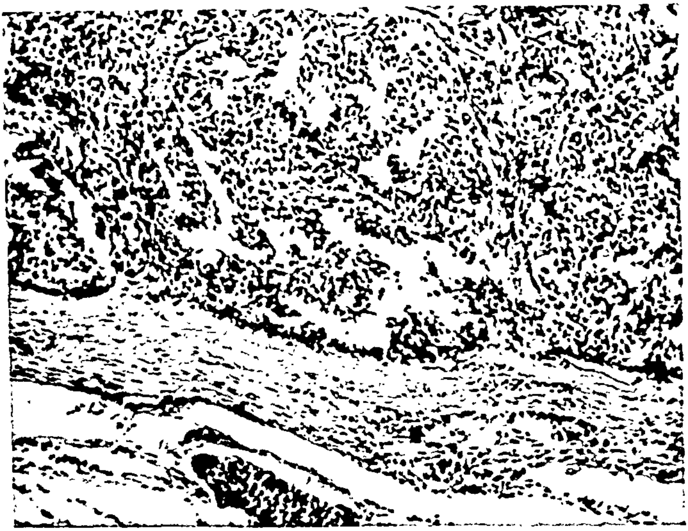

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## SUMMARY

Eight new cases of an unusual type of minute pulmonary neoplasm have been analyzed in respect to their clinical and pathological features. Evidence is presented to show that these tumors are relatively benign in nature, resembling most closely the pattern seen in the carcinoid type of bronchial adenoma. In addition to their histologic structure, other common features of these tumors were their minute size, accidental microscopic discovery, subpleural location, and overwhelming predilection for females. The concomitant occurrence of bronchiectasis in the majority of the recorded cases suggests that this condition may be a factor in the pathogenesis of this atypical type of epithelial cell proliferation.

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# TRANSPLEURAL BRONCHOTOMY

ALFRED GOLDMAN, M.D.
LOS ANGELES, CALIF.

TRANSPLEURAL bronchotomy is the operation of incising or resecting portions of the bronchial walls after making that surgical approach usually used for lobectomy, pneumonectomy, or exploratory thoracotomy. The approach to the pulmonary hilum may be posterolateral,[1-3] or anterolateral.[4, 5] In certain instances the purpose of the operation is to remove by local excision, benign, potentially, or semimalignant bronchial tumors usually of a polypoid nature.[1, 3, 5, 6] In other cases it may be utilized as a bronchoplasty for the relief of bronchial stenosis whether of tuberculous[7] or neoplastic origin. Third, it may be used as a part of exploratory thoracotomy to assist in obtaining tissue for frozen section, or in directly visualizing a tumor especially in the diagnosis of those small malignant bronchial and alveolar tumors whose shadows are likely to be discovered for the first time in roentgenogram surveys.

Reports of bronchoplasty for tuberculous stenosis have been made by Gebauer,[7] but only a few recorded operations have been performed for tumor.[1-3] It is the purpose of this paper to report experiences with five transpleural bronchotomies which were attempted for local excision of tumors of the adenomatous group (Table I).

In Table I are summarized some pertinent data of the results of bronchotomy. To appreciate the surgical problem encountered, a summation of each case is in order.

## CASE REPORTS

CASE 1.—A. B., U.C.H. This operation was started with the idea of performing a left pneumonectomy through a posterolateral incision, since preoperative bronchoscopic removals over a period of six years had shown the recurrent adenoma to be located in the left-stem bronchus. Bronchiectasis was severe in the lower lobe. After freeing numerous dense adhesions from the left lower lobe, the patient's condition was deteriorating, so that it appeared wise to terminate the operation without removal of the upper lobe. After amputating the left lower lobe below the upper lobe take-off, the tumor in the left-stem bronchial wall was totally excised (Fig. 1). The edges of the bronchotomy wound were closed with interrupted fine silk. Postoperative course was smooth. Closed tube drainage of the pleural cavity was established at operation but very little air escaped. The tube was removed on the second postoperative day. Follow-up five years since bronchotomy with left lower lobectomy shows that the patient is well, free of signs of recurrence and suppurative symptoms, despite some bronchiectasis having been proved in the left upper lobe before surgery. (An illustration of the surgical technique used in this case has been previously reported.[8])

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From the Thoracic Surgical Services, Cedars of Lebanon, and Harbor General Branch of the Los Angeles County Hospital.

Read (by title) at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, New Jersey, April 16-18, 1951.

CASE 2.—E. S., C.L.H. This operation was begun as a transpleural bronchotomy through an anterolateral approach. It was expected from the preoperative bronchoscopic studies which had been carried out for treatment of this cylindromatous adenoma for six years before transpleural bronchotomy, that a condition similar to that of Case 1 (Fig. 1) would be found, but instead widespread pleural and mediastinal metastases were discovered. Since both right- and left-stem bronchial walls as well as the carina were extensively infiltrated, complete excision of the tumor for relief of bronchial obstruction was out of the question. The asphyxial symptoms might be relieved by a bronchoplasty, and this was done by incising completely the lower anterolateral wall of the trachea and left-stem bronchus in a longitudinal direction for a distance of 2.5 cm. and suturing with fine silk the cut edges in a horizontal plane so as to widen the orifices of the left-stem bronchus and lower end of the trachea. From this point, until closure of the bronchoplasty was complete, anesthetic oxygenation was carried on through a 22F uretheral catheter inserted into the right-stem bronchus transpleurally. The bronchial incision extended through malignant neoplastic tissue. Closed tube drainage of the pleural cavity was established, but prac-

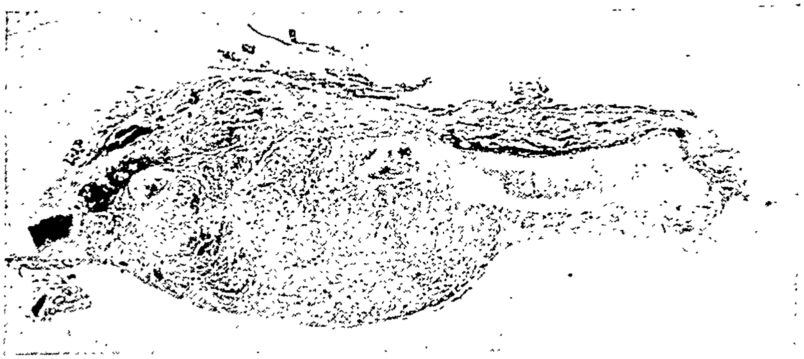


Fig. 1.—Case 1. A.B., U.C.H., woman, aged 38 years, S.P. 46-589. Low-power magnification of surgical specimen of recurrent carcinoid bronchial adenoma removed at bronchotomy. Note that five bronchial cartilages are included in the specimen. The specimen was 3.5 cm. by 1.0 cm. by 1.0 cm.

TABLE I

| CASE | YEAR | SEX | AGE | DIAGNOSIS | RESULT OF BRONCHOTOMY | FOLLOW-UP |
|---|---|---|---|---|---|---|
| 1 A. B. | 1946 | female | 38 | Carcinoid adenoma. Recurrent after bronchoscopic treatment. | Tumor removed with bronchial wall from left stem, 4 cartilages. Left lower lobectomy | Living and well. Five years no recurrence. |
| 2 E. S. | 1947 | male | 58 | Recurrent cylindromatous adenoma with widespread metastasis. | Unable to remove tumor. Bronchoplasty for asphyxia left stem and trachea. | Died 2 months P. O. Metastasis and asphyxia. |
| 3 D. P. | 1949 | female | 14 | Serous glandular adenoma. | Tumor removed left stem. Base cauterized, direct vision. | Living and well 2 years. No recurrence. |
| 4 S. S. | 1949 | female | 45 | Carcinoid adenoma. Recurrent after bronchoscopic treatment. | Tumor removed right stem. Two cartilages. | Living and well, 18 months. |
| 5 B. P. | 1949 | male | 26 | Carcinoid adenoma. Bronchoscopic treatment attempted but failed. | Unable to remove tumor of left lower lobe. Invaded left upper lobe bronchus. Left pneumonectomy. | Living and well, 2 years. |

tically no air escaped, and the tube was removed on the third day. Although the patient obtained partial relief from dyspnea and was able to leave the hospital on the twelfth postoperative day, two months later he succumbed shortly after readmission from asphyxial symptoms. Post-mortem examination disclosed the widespread infiltration of the bronchial and adjacent tracheal walls. Surprisingly, the suture line of the bronchoplasty was well healed. The pathologic findings in this case have been reported previously in the JOURNAL OF THORACIC SURGERY.[8]

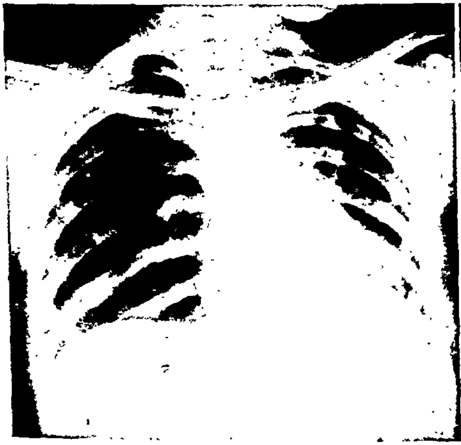


Fig. 2.

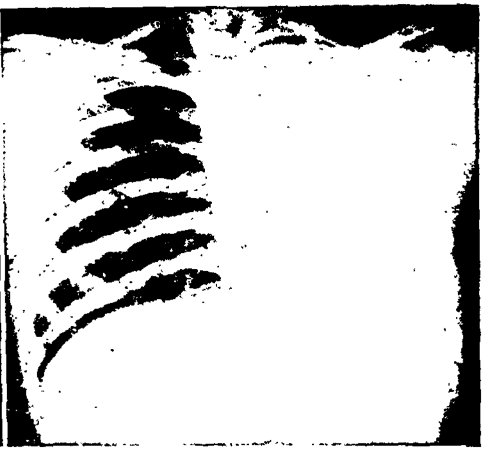


Fig. 3.

Fig. 2.—Case 3. D.P., H.G.H., girl, aged 14 years, Feb. 28, 1949. Roentgenogram of the chest showing abnormalities of the left hilar region at a time when wheezing was prominent and "asthma" was the diagnosis. Left lower lobe atelectasis probably present.

Fig. 3.—Case 3. D.P., H.G.H., girl, aged 14 years, April 15, 1949. Roentgenogram of the chest showing complete atelectasis of left lung.

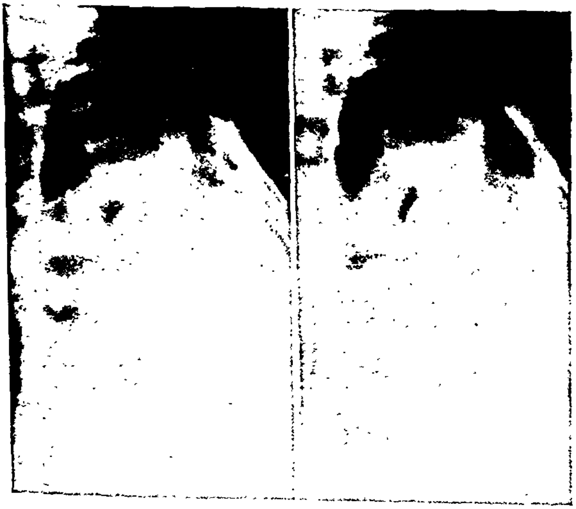


Fig. 4.—Case 3. D.P., H.G.H., girl, aged 14 years, April 16, 1949. Planigram levels 3 and 4 showing complete occlusion of left-stem bronchus from tumor mass extending to carina.

CASE 3.—D. P., H.G.H. From the preoperative radiologic study (Figs. 2, 3, and 4), bronchoscopic findings, and the histologic evaluation of the biopsy, it was felt that a large polypoid tumor belonging to the adenomatous group was obstructing completely the left-stem bronchus, and, although wheezing had been present for more than a year, complete bronchial occlusion was only about one week's duration. It was believed that pulmonary suppuration would not ensue postoperatively if local excision could be utilized without producing bronchial stenosis. Accordingly, a transpleural bronchotomy was performed April 20, 1949, through a posterolateral incision. The left lung was airless and heavy. The posterolateral aspect of the left-stem bronchus and lower trachea were easily exposed below the arch of the aorta. The tumor could be palpated through the bulging bronchial tube, but nowhere was there a visible or palpable sign of the tumor infiltrating beyond the bronchial wall. Adjacent lymph nodes were innocent in gross appearance and frozen sections. The bronchotomy incision extended three centimeters longitudinally from the lower end of the trachea to the take-off of the superior segmental bronchial orifice of the lower lobe. Although most of the incision was through the soft muscular wall of the left-stem bronchus, it was not possible to avoid cutting some cartilaginous rings. The large cystic polypoid tumor (Figs. 5, 6, and 7) mushroomed out of the bronchotomy opening into the pleural cavity so that with gentle traction on the tumor the mesial wall and point of attachment by a stalk 0.5 cm. in diameter could be thoroughly examined. This examination of the interior of the stem bronchus was made easier by the aspiration from the lower lobe and main bronchus of some 30 c.c. of slightly turbid damned-up fluid, more serous than mucous. Careful palpation of the mediastinal outside surface of the mesial wall and subcarinal area failed to disclose any evidence of invasion beyond the cartilages at the point of attachment. Therefore, the stalk was amputated flush with the internal surface of the cartilages including a 2 to 3 mm. rim of bronchial mucosa. Small pieces of adjacent bronchial mucosa were also taken for histological examination. The 1.0 cm. crater left denuded of mucosa was now superficially cauterized with the high frequency endotherm cautery. The bronchotomy incision was closed with one layer of interrupted fine silk; no attempt to reinforce the suture line with mediastinal pleura was made. The anesthetist expanded the

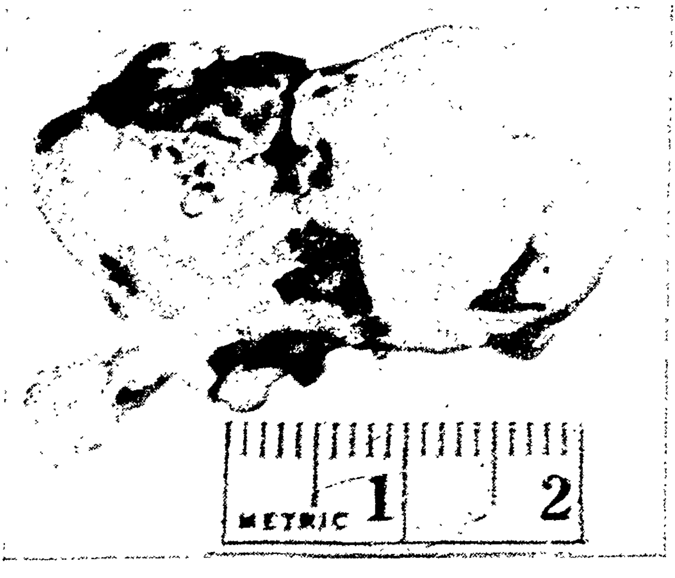


Fig. 5.—Case 3. D.P., H.G. H., S.S. 516-49. Gross surgical specimen removed from left-stem bronchus in toto via transpleural bronchotomy. Note cystic structures and stalk.

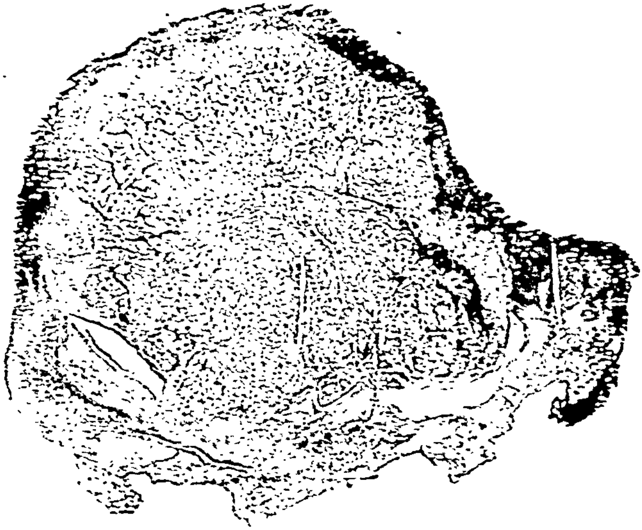


Fig. 6.—Case 3. D.P., H.G.H., S.S. 516-49. Cross section at right angles to long axis of tumor seen in Fig. 5. Low power. Note glandular structure and systic areas and bronchial mucosa covering endobronchial surface of tumor.

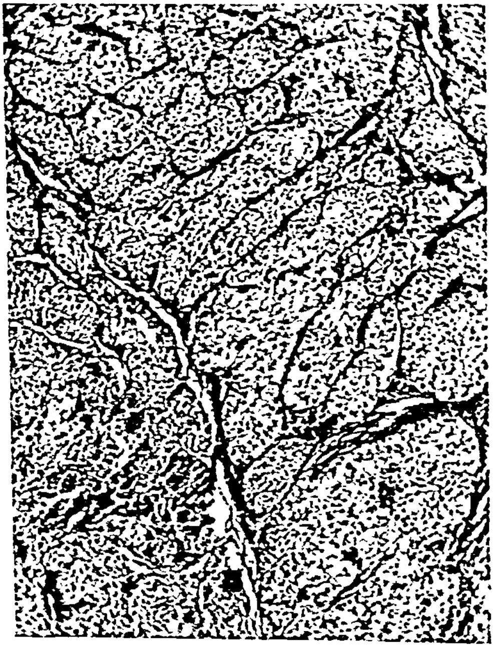


Fig. 7.—Case 3. D.P., H.G.H., S.S. 516-49. High-power view showing glandular structure, numerous delicate connective tissue septa, and clear cell. Tumor probably arose from serous mucous bronchial glands.

lung with some difficulty but no leakage of the bronchotomy opening was observed. Closed tube drainage of the pleural cavity was established. Only a small amount of air escaped through the tube the first day. The tube was removed on the second day.

Re-expansion of the lung was carefully observed with frequent roentgenograms of the chest. Almost complete atelectasis was observed the first two days (Fig. 8). Tracheal aspiration appeared to aid the re-expansion and thoracentesis was necessary. By the third day, only a small amount of fluid remained and the upper lobe was almost completely expanded (Fig. 9). In order to observe the configuration of the bronchi, bronchograms were done one month (Figs. 10*A* and 10*B*) and fourteen months postoperatively (Figs. 11*A* and 11*B*). One month postoperatively, clubbed shaped bronchial dilatations were delineated in the superior segment of the left lower lobe, and cylindrical dilatations in other segments, although at this time the patient had no cough or sputum. One year and two months postoperatively (Figs. 11*A* and 11*B*) the bronchograms fail to delineate any club-shaped bronchiectasis in the superior segment of the left lower lobe and the cylindrical dilatations are also less marked, but are present. She had no febrile episodes during this time, and has missed no time from school. Bronchoscopic examination eighteen months after transpleural bronchotomy shows a normal lumen to the stem bronchus, some black silk is seen covered by transparent mucosa: no signs of suppuration. She has remained well for two years with no sign of recurrence.

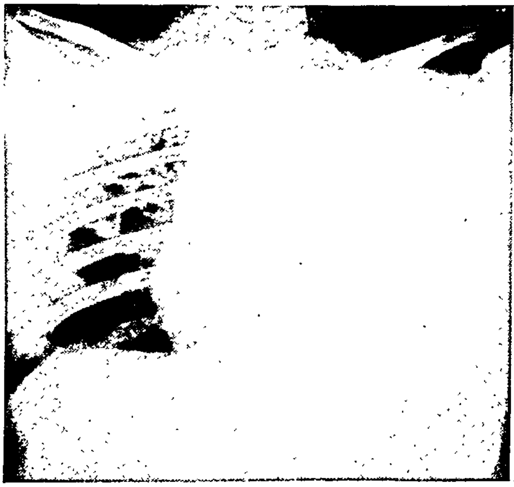


Fig. 8.

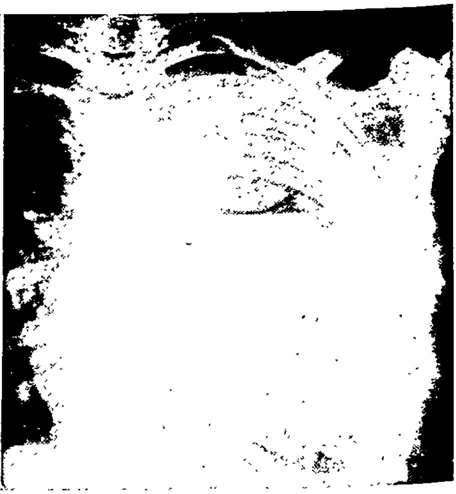


Fig. 9.

Fig. 8.—Case 3. D.P., H.G.H., April 22, 1949. Second day postoperative transpleural bronchotomy. Roentgenogram of the chest showing pleural fluid and atelectasis of the left lung.

Fig. 9.—Case 3. D.P., H.G.H., April 23, 1949. Third day postoperative transpleural bronchotomy. Roentgenogram of chest showing marked clearing of atelectasis and fluid; compare with Fig. 8.

The bronchogram studies one year apart (Figs. 10*A* and *B* and 11*A* and *B*) are significant because they, as well as the bronchoscopic examinations, demonstrate that bronchial stenosis does not result after transpleural bronchotomy with removal and direct cauterization of the base of a polypoid tumor of this type. A cure can be expected because no neoplastic tissue was seen in the biopsies of the mucosa surrounding the base, and because there has been no sign of recurrence for more than two years. Nevertheless, annual bronchoscopic examinations have been advised.

Case 4.—S. S., C.L.H., presented a problem similar to that of Case 1. In a woman of 45 years, a carcinoid adenomatous polypoid tumor had recurred five years after the entire tumor was thought to have been removed bronchoscopically. Roentgenologic and bron-

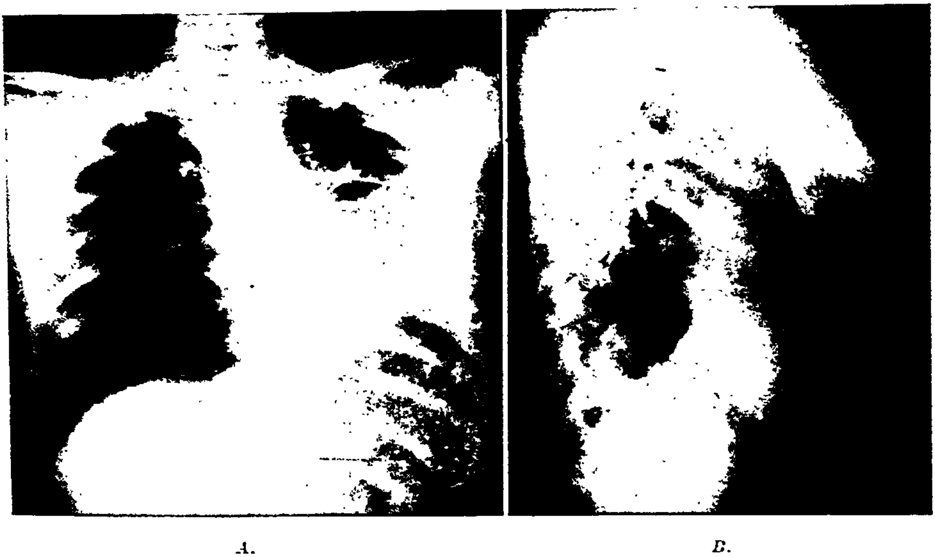


Fig. 10A.—Case 3. D.P., H.G.H., May 16, 1949. Bronchogram one month after transpleural bronchotomy. Posteroanterior view showing club-shaped dilatations in the superior segment of the left lower lobe and cylindrical bronchiectasis in other segments of the left lower lobe.

Fig. 10B.—Case 3. D.P., H.G.H., May 16, 1949. Bronchogram one month after transpleural bronchotomy. Left lateral view showing puddling of lipiodol in the superior segment of left lower lobe.

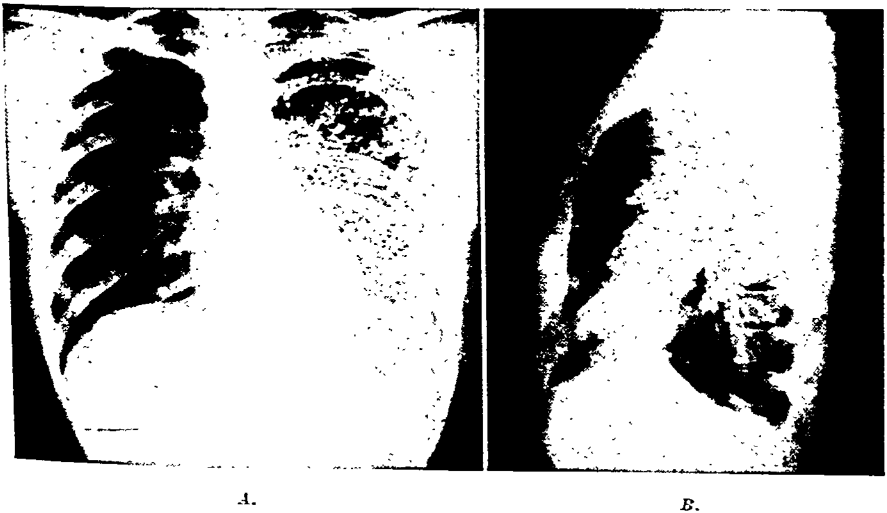
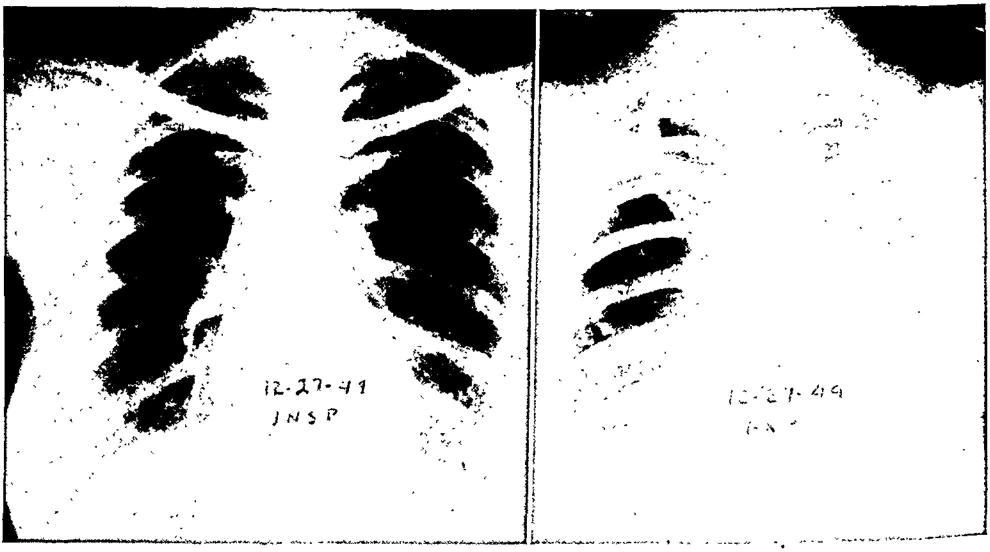


Fig. 11A.—Case 3. D.P. H.G.H., June 15, 1950. Bronchogram one year and two months after transpleural bronchotomy. Compare with Fig. 10A. Posteroanterior view showing failure to delineate club-shaped bronchiectasis seen one year before, and less cylindrical dilatation in other segments. Patient is symptom free.

Fig. 11B.—Case 3. D.P., H.G.H., June 15, 1950. Bronchogram one year and two months after transpleural bronchotomy. Compare with Fig. 10B. Left lateral view. Note again failure to delineate club-shaped bronchiectasis in superior segment of left lower lobe. Compared to Figs. 10A and 10B, Figs. 11A and 11B demonstrate a diminuation in the bronchiectasis following transpleural bronchotomy.

choscopic examinations disclosed that complete obstruction of the right-stem bronchus from this tumor was of only a few days' duration, and, as in the previous case, was associated with taking of bronchoscopic biopsy. In this instance, especially, we were able to observe radiologically the transformation of a right lung with tension emphysema (Figs. 12*A* and 12*B*) into a completely obstructed lung (Figs. 13 and 14). Since this woman had no previous suppurative symptoms, we felt justified to attempt to conserve her lung by performing on Dec. 30, 1949, local excision through transpleural bronchotomy via an anterolateral approach



*A.* *B.*

Fig 12*A* and *B*.—Case 4. S.S., C.L.H., woman, aged 45 years, Dec. 27, 1949. Inspiration and expiration roentgenograms of chest showing shift of mediastinum to contralateral left side and Recurrent carcinoid bronchial adenoma right-stem bronchus, with tension emphysema of the Note, also, tracheal mediastinal displacement and opacity of the homologous right lung.

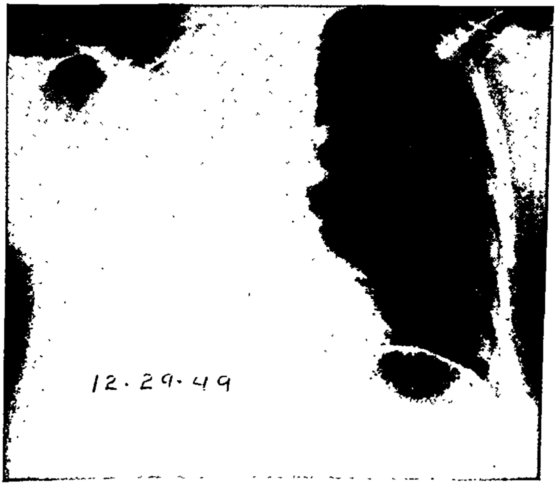


Fig. 13.—Case 4. S.S., C.L.H., Dec. 29, 1949. Twenty-four hours after bronchoscopy. Posteroanterior view roentgenogram of the chest showing complete obstruction and atelectasis of the right lung with mediastinum shifted to the homologous side. Compare with Figs. 12 and 14.

in the third interspace. After entering the pleural cavity, the right lung was found to be heavy and airless. The right-stem bronchus was readily exposed after elevating the lung from its posterior bed and displacing the upper lobe downward. It was difficult to feel the tumor through the bronchial wall, and, as in the previous case, neither mediastinal extension nor adjacent involved lymph nodes were found in frozen sections. The right lung was now replaced in its posterior bed and vena azygos, superior pulmonary vein, and upper-lobe branch of the right pulmonary artery were retracted in order to expose the lower end of the trachea, right upper-lobe bronchus, and anteromedial surface of the right-stem bronchus.

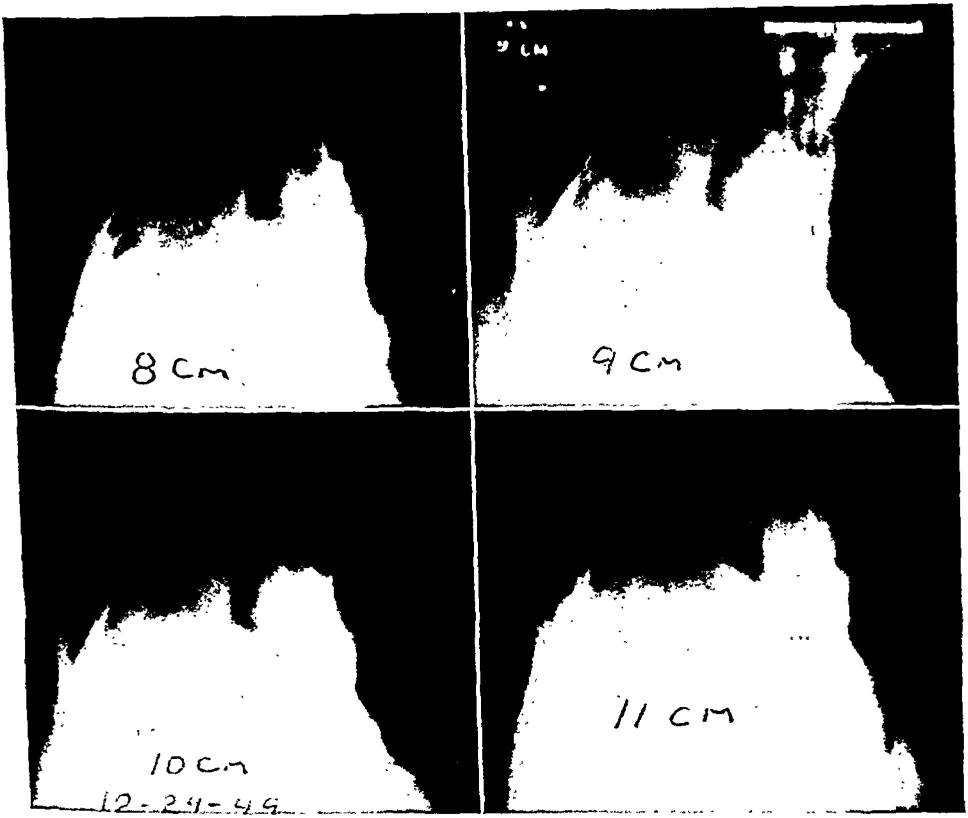


Fig. 14.—Case 4. S.S., C.L.H., Dec. 29, 1949. Planigrams levels 8, 9, 10, and 11, showing the complete obstruction of the right-stem bronchus at the level of the carcina by neoplasm. Note, also, tracheal mediastinal displacement and opacity of the homologous right lung.

An incision was made through the bronchial wall between the cartilages of the anteromesial surface of the bronchus from the orifice of the upper-lobe bronchus to the mediastinal border of the subcarinal surface of the right-stem bronchus. Through this bronchotomy the *interior of the right-stem bronchus was explored and found to contain a fleshy friable* polypoid tumor which was attached to the mesial wall of the right-stem bronchus (Fig. 15). The attachment was sessile and covered an area of approximately one square centimeter. This sessile base involved two large cartilaginous rings (Figs. 15 and 16) which were resected with the tumor. Again bronchial fluid, about 20 c.c. slightly purulent, was encountered. In order to excise the tumor with the bronchial wall (Fig. 17) the bronchotomy incision was continued to completely encircle its sessile attachment. The cartilages were portions of the first and second rings below the carinal cartilage. Closure of this defect

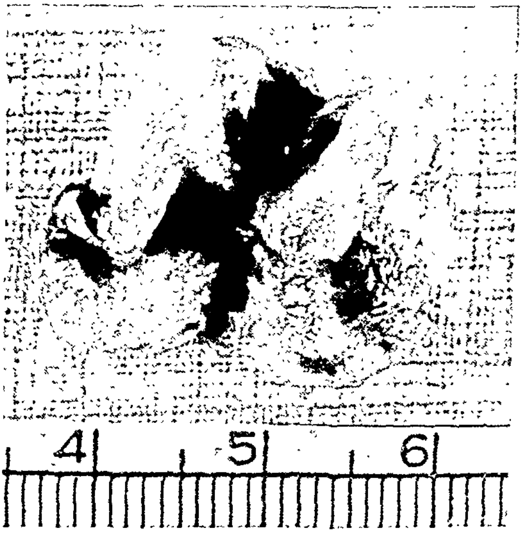


Fig. 15.—Case 4. S.S., C.L.H. Gross surgical specimen of recurrent carcinoid adenoma removed from right-stem bronchus by transpleural bronchotomy.

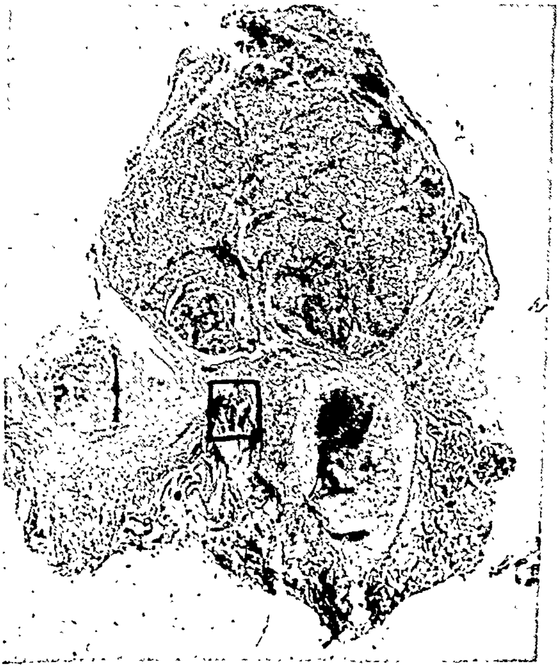


Fig. 16.—Case 4. S.S., C.L.H. Low-power view of cross section of recurrent adenoma shown in Fig. 15. Note the cartilages and bronchial wall resected intact with the endobronchial tumor. See Fig. 17 for high-power of invasive tumor nodule.

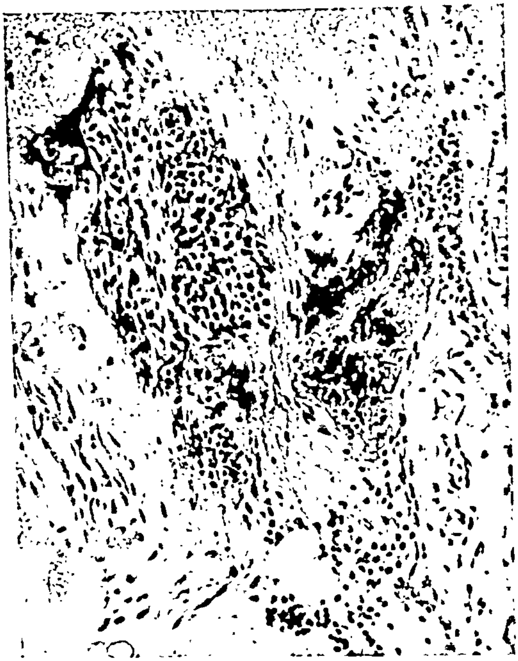


Fig. 17.—Case 4. S.S., C.L.H. High-power view of area beyond the cartilages marked by squares in Fig. 16 showing nodule of adenoma beyond the reach of the bronchoscope. Such nodules probably account for recurrences after bronchoscopic removal.

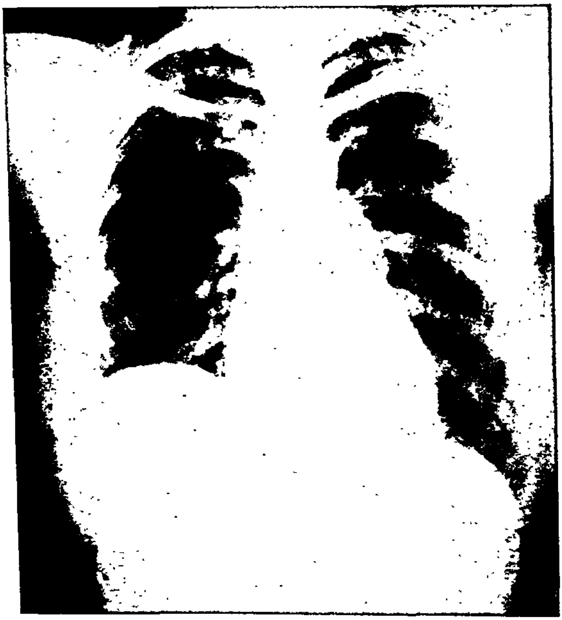


Fig. 18.—Case 5. S.S., C.L.H., April 10, 1950. Roentgenograms of the chest showing slight blunting of the right costophrenic sinus. Satisfactory aeration of right lung three months after transpleural bronchotomy and resection of recurrent bronchial adenoma. Sixteen months after that operation the patient was well, with no signs of recurrence.

was difficult but was managed by displacing the carinal cartilage downward with traction sutures of medium silk that were removed after tying coapting fine silk interrupted sutures. No attempt to cover the defect with pleura was made. Closed tube pleural drainage was established. Postoperatively, there was little leakage of air and the tube was removed on the third day. Expansion of the right lung was satisfactory except for one area of segmental atelectasis. She was discharged on the tenth postoperative day, breathing without wheezing and dyspnea for the first time in months. Three months later a roentgenogram of the chest showed satisfactory aeration of the right lung (Fig. 18) with disappearance of the segmental atelectasis. She has continued work as a saleslady uninterrupted during the past fifteen months. Radiologic examination sixteen months postoperatively discloses essentially the same appearance as in Fig. 18.

CASE 5.—B. P., C.L.H. This white man 26 years of age had had symptoms of mild pulmonary suppuration for more than five years before hemoptysis occurred. Attempts at bronchoscopic removal of a carcinoid adenoma of the left lower lobe failed because of excessive hemorrhage and large size. It was felt that transpleural operation was indicated, and that the operation should be planned so that local excision might be done if the preoperative evaluation proved accurate, i.e., if the tumor had not become too large, was still located in a suitable position where repair of the bronchotomy could be achieved without obstructing segmental orifices; if no metastases were found; if no evidence of irreversible suppuration of the left lower lobe existed; and if the upper-lobe bronchus were not involved. At operation, which was performed through a left posterolateral incision, bronchotomy of the posterior wall of the left lower lobe disclosed an endobronchial portion of the tumor which at first appeared to be amenable to local excision or lobectomy. However, exploration of the mediastinal area about the distal end of the left-stem bronchus demonstrated the iceberglike nature of the tumor, and that the extrabronchial portion invaded from the mediastinal side the wall of the left upper-lobe bronchus. Therefore, a left pneumonectomy was performed. Postoperative convalescense was smooth. Two years postoperatively, a recent examination disclosed him to be well and without evidence of any recurrence.

## DISCUSSION

The experiences described support Eloesser's[1] contentions made in 1940 that transpleural bronchotomy is a feasible conservative method of removing certain "benign" tumors of the bronchus which by reason of their anatomic situations or their doubtful extensions are not amenable to safe and certain bronchoscopic removal. Since 1940, the unpredictable potentially malignant nature of the adenomatous bronchial tumors has been thoroughly proved.[8-13] In spite of this malignant potentiality, slow growth, tendency for encapsulation, and high capacity for being limited by normal tissue environment, such as bronchial mucosa, cartilages and lung parenchyma, make many of the adenomatous bronchial tumors amenable to complete local excision. Furthermore, it appears entirely possible to differentiate those tumors which are amenable to complete local excision via transpleural bronchotomy by the aid of clinical studies before operation[10-13] and by further information obtained at the time of operation. This observer emphatically advises against bronchotomy in every case of adenomatous bronchial tumor, and, of his forty cases, this paper contains the sum total of his experiences with bronchotomy in this type of neoplasm. But, our past knowledge is based on adenomas that were of long duration often accompanied by regional lymph node metastases, marked pneumonitis, bronchiectasis, abscess, and pleuritis, where pulmonary resection must

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# THE NATURAL HISTORY OF SO-CALLED TUBERCULOMAS

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THE term "tuberculoma" of the lung is applied to solid caseous foci, representing undrained tuberculous abscesses filled with inspissated caseous material, from which tubercle bacilli can be recovered in large numbers. The term has also been applied on occasions to other pulmonary lesions.[1, 2]

Persons with such lesions complain mainly of feeling "under the weather" rather than of any specifically respiratory symptoms. Physical signs are absent, the sputum is negative, and the lesion is found only on radiologic examination, and is often detected more easily on screening than with a film, since it may be hidden behind a rib or the scapula. The lesion is roughly spherical in shape, from ½ to 3 cm. in diameter, with sharply defined borders, and fairly uniform density. Smaller daughter foci may be seen near to the parent focus. The diagnosis of a solid focus of tuberculous caseous material is often a presumptive one determined solely on interpretation of the radiologic appearances, and other conditions have to be considered such as primary and secondary neoplasms, cysts, abscesses, and encapsulated interlobar empyemas.

In view of the paucity of symptoms and the circumscribed appearance of the lesion, patients with "tuberculomas" are frequently kept under observation. In others, where a pneumothorax has been induced, the focus has been found unchanged when the lung has been allowed to re-expand. Now it is recommended that the proper treatment of such solid caseous foci is resection of the affected segment of the lung. Such treatment seems drastic if these lesions run a benign course and carry a favorable prognosis.

In an attempt to elucidate this problem, eighteen patients, with a total of twenty-three "tuberculomas," have been found who have been kept under observation. Two patients had two solid caseous foci in one lung, and another had three foci in one lung and one in the other lung. The course of these "tuberculomas" was as follows:

*Calcified.*—Four foci. Observed 15, 22, 54, and 148 months.

*No change.*—Seven foci. Observed 12, 12, 16, 24, 89, 89, and 89 months.

*Enlarged.*—Five foci. Observed 20, 22, 22, 24, and 50 months.

*Cavitated.*—Four foci. Observed 5, 23, 89, and 90 months.

*Cavitated with Bronchogenic Spread.*—Three foci. Observed 30, 38, and 51 months.

It appears from these figures that "tuberculomas" are relatively indolent lesions. Even when cavitation occurred, in four of seven cases bronchogenic

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to relieve asphyxiating malignant obstruction. In the fifth case, the pneumonectomy was necessary because widespread bronchial infiltration had already occurred. Conservation of lung and complete local excision in the carefully selected case is stressed.

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# LOBECTOMY AND PNEUMONECTOMY IN TUBERCULOSIS

## A Report of 54 Cases

L. Johansson, M.L., and L. G. Uggla, M.L.
Stockholm, Sweden

THE first tentative attempts at pulmonary resection in tuberculosis therapy were made in the last two decades of the nineteenth century. But it was not until the third and, in particular, the fourth decades of this century that improved diagnostic technique and chemotherapy permitted a more accurate evaluation of the problems involved in the treatment of pulmonary tuberculosis by resection of the affected tissue. As a natural consequence of the rapid progress of thoracic surgery, it is now possible to study the results in large series of tuberculous patients treated with this procedure. The foremost names in the Anglo-American literature are those of Overholt, Bailey, Sweet, etc. Among European writers, Klinkenberg has reported a large series of resection cases. The Swedish material hitherto published (Crafoord, Rudström, Wulff) was rather small and the observation period was relatively short.

The first of our patients has been observed for fifteen years, and during these years an increasing number of patients have been treated with resection. We consider, therefore, that the time is now opportune for reporting the results of treatment in fifty-four patients who underwent pulmonary resection at Sabbatsberg Hospital in Stockholm during the period 1935-1950.

To obtain a survey of the results in earlier studies, a table has been compiled showing findings from investigations made before the introduction of chemotherapy and from recently published papers concerning operations carried out with the protection of chemotherapy (para-aminosalicylic acid [PAS] and streptomycin). A number of data were obtained through personal communication with Dr. Swirenga (Holland).

No distinction has been made in the table between "good" and "poor" surgical risks. The mortality and complications rates were high in the early series, and were considerably reduced by the advent of chemotherapy. In addition to the beneficial action of the drugs, the patients in the later studies had the advantage of all the advances in surgical and anesthetic technique and postoperative therapy.

The patients who comprised the clinical material in this study all underwent operation at Sabbatsberg Hospital. Rather more than half of them (twenty-nine) received pre- and postoperative care at Söderby Hospital. The others were referred to Sabbatsberg by sanatorium physicians in the provinces. As the surgical procedures were throughout the responsibility of the same surgeon (Prof. C. Crafoord), a continuity was created which was important in the assess-

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From the Surgical Department of Sabbatsberg Hospital, Stockholm, Head: Professor Clarence Crafoord; and Söderby Hospital. Head: Dr. Med. G. Birath and T. Bruce.

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dissemination of disease did not ensue. In only four, however, did definite healing occur, while in twelve the pathologic process continued.

## SUMMARY

The results of observation of twenty-three so-called tuberculomas are reported. Altogether three cavitated with bronchogenic spread, four cavitated only, and five enlarged.

Thus resection of the affected segment of the lung, since it carries practically no mortality and does not entail the sacrifice of any appreciable amount of lung tissue, seems to be the treatment of choice for these solid lesions. Especially is this so when the difficulty in making a differential diagnosis from peripheral carcinoma is borne in mind. A correct diagnosis of carcinoma may only become evident at a stage when curative treatment for this condition has become impossible.

I wish to express my appreciation to Mr. F. Ronald Edwards for his interest, advice, and criticism.

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ment and planning of the interventions. On the other hand, the preoperative and late postoperative treatment was inevitably subject to variations from the fact that the patients were hospitalized in different institutions.

The operative procedures were as follows: 18 lobectomies, 3 lobectomies combined with segmental resection, 1 segmental resection, and 32 pneumonectomies. The last-named included 7 cases of pleuro-pneumonectomy, an intervention which undoubtedly involves increased risk of hemorrhage and shock. The predominance of women (thirty-three) may possibly be ascribed to the higher incidence of bronchial tuberculosis in females.

TABLE I.

| AUTHOR | NUMBER OF CASES |  | TOTAL DEATHS |  | POST-OPERATIVE MORTALITY |  | SPREADS |  |
|---|---|---|---|---|---|---|---|---|
|  | NO SPECIFIC CHEMO-THERAPY | SPECIFIC CHEMO-THERAPY | NUMBER | PER CENT | NUMBER | PER CENT | NUMBER | PER CENT |
| Overholt et al. | 196 |  | 46 | 24 | 27 | 14 | 54 | 28 |
| Sweet | 63 |  | 28 | 44 | 9 | 16 | 20 | 32 |
| Clagett et al. | 29 |  | 4 | 14 | 2 | 7 | 8 | 28 |
| Bailey et al. | 100 | 100 | 37<br>16 | 37<br>16 | 27<br>16 | 27<br>16 | 15<br>1 | 15<br>1 |
| Moore et al. |  | 74 | 6 | 8 | 6 | 8 | 3 | 4 |
| Day et al. | 202 |  | 25 | 12 | 14 | 7 | 25 | 12 |
| Himmelstein et al. | 73 |  | 15 | 21 | 12 | 16 |  |  |
| Jones et al. | 35 | 88 | 10<br>5 | 29<br>6 | 8<br>5 | 23<br>6 | 7<br>4 | 20<br>5 |
| Munz et al. |  | 53 | 4 | 8 | 4 | 8 | 1 | 2 |
| Swierenga |  | 227 |  |  | 9 | 4 |  |  |

Those patients who received no specific chemotherapy (twenty-three in number) were for the most part operated on before 1948. The youngest of the series was 8 years old and the oldest 63 years of age. None of the patients had been observed for a shorter period than one year after the disease was diagnosed. The great majority had been followed for many years, and during that time had received sanatorium care when such was necessary. In the patients classed as having bilateral disease, the contralateral process was practically always estimated to be clinically inactive. No patient had permanent contralateral collapse, but two had earlier contralateral artificial pneumothorax. The number of pneumonectomies performed during the different periods of the investigation was: 1935-1942; six; 1943-1947, eight; 1948-1949, eight; and up to September, 1950, ten. Corresponding figures for lobectomies were: zero, five, nine and eight.

Throughout the two tables of indications distinction has been made between patients who had previously received active therapy either in the form of permanent collapse (thoracoplasty) or temporary collapse (pneumothorax or pneumoperitoneum with phrenic nerve interruption), and cases where resection was the first active measure. The indications for operation were not notably divergent from those reported in the Swedish and foreign literature. Thus one group consisted of fourteen patients with residual cavities after thoracoplasty or revision thoracoplasty or with cavities so localized that collapse therapy was unlikely to be effective; these cavities were usually situated in the lower lobe. Another important indication was bronchostenosis secondary to tuberculosis with intermittent or constant retention symptoms and with positive sputum (seventeen patients). This group included some cases where the changes peripheral to the stenosis were so marked that the pulmonary parenchyma was more or less destroyed. A third group comprised 6 patients with bronchiectasis secondary to tuberculosis and persistent and positive sputum. Where sputum conversion can be obtained, these patients should be submitted to surgery on the same indications as for nontuberculous bronchiectasis. In 2 patients who underwent operation for nonspecific bronchiectasis, tuberculosis was revealed by microscopic examination. Those cases (seven in number) where earlier therapy had resulted in fistula formation and empyema presented great difficulties. Pleuro-pneumonectomy was found to be the only and final resort for such patients in our study. Extensive unilateral tuberculosis constituted the indication for surgery in five patients. The series also includes three cases of tuberculoma, two of which were thought to be tumors. More unique than the above-named common indications is a case of arteriobronchial fistula which has previously been described by Crafoord.

When judging the indications in all these patients, the appearance of the bronchial tree was considered to be as important as the extent of the parenchymatous lesions. No less than twenty-seven of the fifty-four patients were found to have definite bronchial tuberculosis. We have, however, always endeavored by local and general therapy to convert the bronchial lesions to inactivity before operation.

Prior to 1948 no patient had received specific chemotherapy. To begin with, chemotherapy was given only on special indications: preoperative chemotherapy was reserved for "poor risks" and postoperative for complications. Gradually, as the drugs became more easily procurable, a more methodic system of therapy was adopted. As far as possible PAS was given preoperatively (more recently some patients have received thiosemicarbazone), and streptomycin was reserved until the time of operation so as to avoid early development of bacillary resistance.

There was, however, a group of patients for whom the initial prospect was considered favorable for thoracoplasty but the course of events necessitated the use of intensive chemotherapy: in these patients resistance to streptomycin had developed before resection was performed. In a few cases of extensive disease, thorough medication with all available chemotherapeutics failed to make the patient fit for collapse therapy; primary resection was then carried out but bacillary resistance had already developed.

No uniform schedule of therapy can be given as the duration of treatment varied very widely in the different patients. Preoperative PAS medication up to five and six months in length was not uncommon. Streptomycin was sometimes added in short courses of ten to fourteen days during PAS therapy. During the last year, streptomycin has always been given in combination with PAS so as to delay bacillary resistance. The usual dosage of PAS was 14 Gm. granulate (70 per cent PAS) daily in 4 divided doses. Streptomycin was given in a dosage of 1 Gm. daily; dihydrostreptomycin has been used since 1949. Thiosemicarbazone was given in the usual increasing dosage up to a maximum of 0.05 Gm. four times daily. PAS and streptomycin were also used locally, particularly in the cases complicated by empyema and fistula formation. Systematic and regular sensitivity tests were, unfortunately, not carried out. In patients with copious sputum containing a variety of bacteria, sulfanilamides and penicillin were added to the preoperative therapy, and were often combined with inhalation therapy. Depending on the clinical findings, streptomycin was continued for two weeks to one month after operation, and PAS and thiosemicarbazone for at least another month.

Preoperative bronchoscopy was performed in all but seven patients. Analysis of cardiorespiratory function has been carried out on an increasing scale during the last few years. The investigations included determination of total capacity, bronchospirometry, registration of the pressure in the pulmonary artery, cardio-angiography, and estimation of oxygen saturation at rest and during exercise.

The plastic surgery procedures which preceded pneumonectomy and lobectomy were carried out with the aid of local anesthesia. For resection operations, intratracheal anesthesia with spiropulsator respiration was used. Surgery was always performed with the patient in the lateral position. In cases with copious sputum, or where the release of secretions during operation was anticipated, the bronchus was sealed by tamponading. For the last year a double-lumen catheter devised by Carlens has been used. With this technique, anesthesia can be administered to each lung through a separate channel. This is highly advantageous when it is necessary to keep the lungs free of secretion and prevent aspiration from the diseased lung. During operation intravenous drip infusions, mainly blood, were given. In this way, protracted and sometimes difficult dissections, e.g., in pleuro-pneumonectomy, could be performed with the minimum of risk. Bronchoscopic aspiration was carried out as a routine immediately after operation. Apart from the specific chemotherapy, the postoperative management of these cases does not differ from that of other intrathoracic conditions with reference to fluid balance, oxygen administration, chest aspiration and routine roentgenograms.

The complications which may follow operation arise from the specific infection and/or the trauma inflicted by surgery on blood vessels, air passages, and pulmonary parenchyma. To the latter group belong the three pneumonectomies which terminated in fatal surgical shock syndrome. In the survey of complications, the clinical material has been divided into cases with and without specific chemotherapy. Among the lobectomy patients, strangely enough, all the

TABLE II. CLINICAL MATERIAL

| | LOBECTOMIES | PNEUMONECTOMIES | |
|---|---|---|---|
| Total patients | 22* | 32† | 54 |
| Men | 8 | 13 | 21 |
| Women | 14 | 19 | 33 |
| Age | 17 to 63 years | 8 to 58 years | |
| Average age | 34 years | 34 years | |
| Duration of disease | 1 to 23 years | 1 to 24 years | |
| Average duration | 8 years | 8 years | |
| No specific chemotherapy | 6 | 17 | 23 |
| Specific chemotherapy | 16 | 15 | 31 |
| Unilateral disease | 14 | 22 | 36 |
| Bilateral disease | 8 | 10 | 18 |
| Previous contralateral collapse (pneumothorax) | 1 | 1 | 2 |

*Including three cases of combined lobectomy and segmental resection and one segmental resection.

†Including seven pleuro-pneumonectomies.

TABLE III. LOBECTOMIES

| INDICATIONS | TOTAL | PREVIOUS ACTIVE THERAPY | | NO PREVIOUS ACTIVE THERAPY | BRONCHIAL LESIONS |
|---|---|---|---|---|---|
| | | PERMANENT COLLAPSE | TEMPORARY COLLAPSE | | |
| Cavities: | | | | | |
| Upper lobe | 4 | 4 | 0 | 0 | 1 |
| Lower lobe | 7 | 1 | 3 + 1* | 2 | 2 |
| Upper and lower lobes | 1 | 0 | 0 | 1 | 1 |
| Tuberculous bronchiectasis | 4 | 2 | 1* | 1 | 1 |
| Tuberculoma | 1 | 0 | 0 | 1 | 0 |
| Suspected tumors† | 3 | 0 | 0 | 3 | 0 |
| Non-specific bronchiectasis | 2 | 0 | 0 | 2 | 0 |
| Total | 22 | 7 | 5 | 10 | 5 |

*Prophylactic pre-lobectomy thoracoplasty.

†Two tuberculomas, one pulmonary tuberculosis + bronchiectasis.

Three patients not examined by bronchoscopy.

TABLE IV. PNEUMONECTOMIES

| INDICATIONS | TOTAL | PREVIOUS ACTIVE THERAPY | | NO PREVIOUS ACTIVE THERAPY | BRONCHIAL LESIONS |
|---|---|---|---|---|---|
| | | PERMANENT COLLAPSE | TEMPORARY COLLAPSE | | |
| Failed cavity closure | 2 | 1 | 1 | 0 | 0 |
| Bronchostenosis with bronchiectasis | 17 | 11 | 3 | 3 | 17 |
| Extensive unilateral lesions | 5 | 0 | 2 | 3 | 1 |
| Empyema with pleurocutaneous fistula | 2 | 2 | 0 | 0 | 1 |
| Residual cavity + empyema + bronchial fistula | 4 | 4 | 0 | 0 | 1 |
| Empyema + bronchial fistula | 1 | 0 | 1 | 0 | 1 |
| Arteriobronchial fistula | 1 | 0 | 0 | 1 | 1 |
| Total | 32 | 18 | 7 | 7 | 22 |

Four patients not examined by bronchoscopy.

TABLE V. POSTOPERATIVE COMPLICATIONS

| POSTOPERATIVE COMPLICATIONS | PNEUMONECTOMIES |  | LOBECTOMIES |  |
|---|---|---|---|---|
|  | WITHOUT CHEMOTHERAPY | WITH CHEMOTHERAPY | WITHOUT CHEMOTHERAPY | WITH CHEMOTHERAPY |
| Contralateral spread | 4 | 1 | 0 | 1 |
| Ipsilateral spread | 0 | 0 | 0 | 1 |
| Bilateral spread | 0 | 0 | 0 | 1 |
| Tuberculous empyema + bronchial fistula | 0 | 0 | 0 | 2 |
| Tuberculous wound fistula | 0 | 1 | 0 | 1 |
| Tuberculosis of bronchial stump | 1 | 2 | 0 | 0 |
| Staphylococcal empyema | 1 | 1 | 0 | 0 |
| Nonspecific wound infection | 0 | 1 | 0 | 0 |
| Transient atelectasis | 0 | 0 | 0 | 2 |
| Postoperative deaths | 6 | 0 | 0 | 1 |
| LATE COMPLICATIONS |  |  |  |  |
| Contralateral spread | 0 | 0 | 0 | 1 |
| Ipsilateral spread | 0 | 0 | 0 | 1 |
| Bilateral spread | 0 | 0 | 1 | 0 |

TABLE VI. PRESENT STATUS

| TIME OF OPERATION | NUMBER OF PATIENTS | CLINICALLY WELL SPUTUM NEGATIVE | CARDIORESPIRATORY INSUFFICIENCY | ACTIVE OR POSSIBLY ACTIVE DISEASE* SPUTUM |  | DEAD |
|---|---|---|---|---|---|---|
|  |  |  |  | POS. | NEG. |  |
| *26 Pneumonectomies* |  |  |  |  |  |  |
| 1935-1942 | (6) 6 | 5 | 3 | 0 | 0 | 1 |
| 1943-1947 | (8) 3 | 2 | 0 | 1 | 0 | 0 |
| 1948-1949 | (8) 7 | 7 | 2 | 0 | 0 | 0 |
| 1950 | (10) 10 | 8 | 0 | 1 | 1 | 0 |
| Total | (32) 26 | 22 | 5 | 2 | 1 | 1 |
|  |  |  |  | 3 |  |  |
| *21 Lobectomies* |  |  |  |  |  |  |
| 1935-1942 | 0 | – | – |  |  | – |
| 1943-1947 | (5) 5 | 2 | 0 | 1 | 0 | 1 |
| 1948-1949 | (9) 9 | 7 | 0 | 0 | 1 | 0 |
| 1950 | (8) 7 | 5 | 0 | 1 | 1 | 0 |
| Total | (22) 21 | 14 | 0 | 2 | 2 | 1 |
|  |  |  |  | 4 |  |  |

In the lobectomy group no information is available in two cases.
The figures in parentheses represent the original number of patients in the study.
*In the sputum-negative patients in this group there is manifest or suspected roentgenographic activity.

complications are registered in the chemotherapy group. This, however, is explained by the fact that only six of the twenty-two lobectomy patients received no chemotherapy. Moreover, the complications of ipsilateral and bilateral spread and the two cases of empyema are accounted for by only two patients; one of these had segmental resection, and the other segmental resection with lobectomy. In the latter patient the tubercle bacilli were almost entirely resistant to both PAS and streptomycin. In the case where a tuberculous wound fistula developed, all available chemotherapy had also been fully utilized prior to operation. Contralateral spread occurred in one patient and transient atelectasis in two.

In the pneumonectomy patients, spread occurred in four who had received chemotherapy and in one who had not. Despite the high incidence of bronchial lesions, tuberculosis of the bronchial stump appeared in only three patients. Two of these patients had received chemotherapy. Staphylococcal empyema developed in one treated and one untreated patient. Nonspecific wound infection occurred in one treated patient.

Of the postoperative deaths, three have already been reported as due to surgical shock. None of the three remaining patients who died after pneumonectomy had received chemotherapy: they developed extensive contralateral spreads which could not be controlled and became caseous. In one lobectomy patient, fistula formed and empyema developed with bilateral spread and finally hemoptysis and death. Of the seven patients who died, four must be regarded as poor surgical risks.

Twenty-one lobectomy patients thus survived operation, but one of them died of cardiac disease three years later. Two are known to be alive but no information is obtainable as to their present condition. Fourteen show no clinical or roentgenological signs of active tuberculosis and four are considered as still having active disease. In two of these four patients no thoracoplasty was performed over the excised lobe. Overdistention of the remaining pulmonary parenchyma may therefore have been a significant factor in the occurrence of spread. It is interesting, in this connection, to note that a number of patients who during recent years underwent lobectomy or pneumonectomy for other nontuberculous conditions, developed tuberculous lesions or exacerbations in the overdistended parenchyma. None of these patients was observed to have cardiorespiratory insufficiency.

Of the twenty-six patients who survived pneumonectomy, one died of staphylococcal empyema two years after surgery. Of those now alive, twenty-two show no clinical or roentgenologic evidence of active disease, and three are classed as "not healed." Two of these three patients have positive sputum. The five cases of cardiorespiratory insufficiency are also registered in the pneumonectomy group. This condition is not verified in any case, but is recorded on the basis of information supplied by the patient himself. The patients classified as "not healed" have developed spreads or are assessed as uncertain on other grounds. They are at present receiving temporary collapse therapy or sanatorium care.

During recent years, one or more sputum cultures or gastric washings have always been examined for *M. tuberculosis* after operation. Unfortunately, no uniform plan has been followed. With regard to those patients who underwent operation a number of years ago, reliance has had to be placed to a certain extent on routine direct smears or sputum concentrates.

## CONCLUSIONS

Specific chemotherapy has made an important contribution to the good results obtained. Patients previously considered to have a hopeless prognosis were converted to a more favorable condition for further therapy. This means that an ever-increasing number of patients is prepared for surgery. Chemotherapy must, however, be employed in such a way that its action is not dissipated at the time of the planned operation. For this it is important that there should be close cooperation between the sanatorium physician and the thoracic surgeon in planning the course of therapy at an early stage. Resection of pulmonary parenchyma is followed by overdistention and emphysema of the remaining pulmonary tissue, and the risk of spread is thereby increased. It has therefore been considered advisable to perform thoracoplasty over the site of the excised tissue. Few exceptions have been made to this rule, and these have resulted in one or two serious reverses. In recent years we have performed primary resection only in those cases where preliminary thoracoplasty was considered to be precluded by the toxicity of the disease and the patient's poor general condition. Thoracoplasty was performed when the patient's condition had sufficiently recovered.

Segmental resection should be reserved for well-defined and localized lesions with little or no activity. Otherwise there is considerable risk of fistula formation, empyema, and spread of the disease.

Careful testing of function should precede operation in every case where it is important to conserve functioning parenchyma and avoid a more disabling cardiorespiratory insufficiency.

In the light of our present experience, resection therapy in pulmonary tuberculosis appears to be a necessary and advisable complement to collapse therapy in suitable cases. Pulmonary resection does not involve a greater surgical mortality or risk of complications than surgical collapse therapy in general. It may be directly lifesaving in cases of unilateral disease with grave general toxicity where other methods of treatment are out of the question: stenosis of the main bronchus with complete pulmonary destruction constitutes such an indication.

## SUMMARY

A report is presented of a series of resection operations carried out on patients with pulmonary tuberculosis at Sabbatsberg Hospital, Stockholm, during the period 1935-1950. The series comprised thirty-two pneumonectomies, twenty lobectomies, and two lobectomies combined with segmental resection. The main indications were: (1) failure of collapse therapy to effect cavity closure; (2) pulmonary lesions secondary to stenotic bronchial tuberculosis; and (3) extensive parenchymatous lesions.

Prior to 1948, nineteen patients underwent operation without the protection of specific antimicrobial agents. There were seven deaths in this group, six of which occurred in the immediate postoperative period.

The thirty-five patients submitted to operation since the beginning of 1948 received specific medication. One of these patients died in the immediate postoperative period, and one later.

The present condition of the surviving patients is reviewed.

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# ANGIOGRAPHIC STUDIES AFTER PULMONARY RESECTION

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During the last few years the field of thoracic surgery has developed new techniques on pulmonary resection, from segmental resection to pneumonectomy; at the same time both surgeons and physiologists have become more interested in the knowledge of the functional state of the rest of the pulmonary tissue, as the patient requires a correct hematosis to continue living.

Many investigators have been concerned with the problems of research including functional, spirometric, and bronchospirometric studies after the therapeutic methods which reduce the field of hematosis (collapse therapy, pneumonectomy, etc.) have been used. Cournand and Richards,[1] in 1941, stressed the effect of various types of collapse therapy upon cardiopulmonary function, and observed a decrease in the maximal breathing capacity after exercise. The same observations were made by Cicero[2] in his receptional thesis in respect to extrapleural pneumothorax. Cournand and Riley[3] in 1950 reported sixteen cases, seven of which were seen shortly after pneumonectomy and nine long after they had been subjected to this therapeutic procedure; in the first group there was a less marked decrease in the maximal breathing capacity after exercise than in the second. Burnett and Long[4] reported a decrease in the maximal breathing capacity in twenty-four patients after they had been pneumonectomized. Long and Norris[5] performed bronchospirometric studies in bronchiectasis before and after lobectomy in sixty-four patients, in some cases there was no variation in the maximal breathing capacity, while in other cases there was an increase. Taylor, Roos, and associates,[6] in their paper on "Respiratory and Circulatory Studies in Patients after Bilateral Lobectomy" reported ten cases in which there was a decrease, both in the maximal breathing capacity and in the oxygen saturation. Peters, Roos, and associates[7] studied ten children after they had been pneumonectomized, and observed that both the oxygen saturation and the maximal breathing capacity gave almost normal values, and concluded that there is no relation between the time in which the operation was performed and the functional state of the rest of the pulmonary tissue, as long as adequate training was carried out in these patients tending to maintain the mobility of the diaphragm and mediastinum.

In most of the cases roentgenologic studies of the chest some time after lobectomy are unable to give information as to the functional state of that lung in as much as the other lobes tend to re-expand and fill the place left in the thoracic cavity, the diaphragm returns to its normal position; in some cases the shadow cast by the lung parenchyma may be less dense due to overdistention.

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In young patients and in those in whom there are no postoperative complications such as broncho-pleural fistulas or empyemas, the roentgenogram of the chest seems to be that of a normal subject.

The visualization of the pulmonary vessels may give some information as to the functional state of the pulmonary tissue left and Neuhof and Nabatoff have already used angiography[8] to study the form and function of the remaining lung after pneumonectomy. Keil and Schissel[9] using the same method were able to make the differential diagnosis of unresolved pneumonia and bronchiogenic carcinoma.

The technique of angiography which we used is the one first described by Dr. Celis which consists in the introduction of a Nelaton's type of catheter through the external jugular vein in the neck down to the right cardiac chambers or stem of the pulmonary artery, injecting 30 or 40 c.c. of 80 per cent Nosylan and taking a roentgenogram half a second after the opaque substance has been injected, thus obtaining the image of the stem of the pulmonary artery and its main branches.

In a normal person being subjected to angiographic study in the antero-posterior position, the stem of the pulmonary artery arising from the conus arteriosus of the right ventricle extends obliquely upward and backward, and after a short course is divided into right and left branches. The first, longer and larger than the second, runs horizontally to the right to the root of the right lung. The left branch passes backward and slightly outward, lying axially to the frontal plane. The right branch of the pulmonary artery gives off its first branch to the upper lobe at the root of the right lung where it bends, showing a curve concave externally thus making it a descending artery, and constitutes the blood supply of the apical and anterior segments; the branch of the posterior segment arising from the stem runs outward; the two branches which supply the middle lobe arise somewhat lower and inward; the branch which supplies the dorsal segment arises outward, while the basal branches are given off downward. The left branch of the pulmonary artery, shorter and somewhat smaller than the right, has a more atypical distribution, most of the anatomists consider from three to seven branches for the upper lobe, while it is not possible to determine the number from an angiographic point of view. In some cases the main branch shows only a common stem, in other cases several secondary branches arise directly from the main stem; immediately downward and outward is the branch to the lingular lobe, the branch of the dorsal segment arises in front and inward, and the basal branches arise downward. All of these arterial branches give off more branches as they run to the periphery, thus originating a "rich and complete circulation."[11]

In the lateral position the same distribution may be seen, only that the shadows cast by both pulmonary arteries are superimposed, thus making it difficult to identify the branches of each pulmonary artery.

The angiographic study gives valuable information as to the functional state of the pulmonary vessels and mediastinum:

1. Position of the pulmonary artery; in pneumonectomized patients we have observed that the pulmonary artery and mediastinum are displaced toward

the operated side if no method, such as thoracoplasty or filling of the cavity with plastic material, is carried out to prevent such a displacement which causes overdistention of the other lung, and consequently a state of respiratory insufficiency.

2. Section of the corresponding arterial branch; this is purely anatomic and is manifested by a sudden stop of the opaque substance at the place of the sectioned branch.

3. Separation of the arterial branches; this is due to the cavity left and overdistention of the remaining lobes or lung, the arterial branches seem to be less and more separated than normally, indicating a poor blood supply and consequent defect in the hematosis; the angiogram after pulmonary resection differs from that in pulmonary emphysema in which there is a diminished blood supply at the periphery.

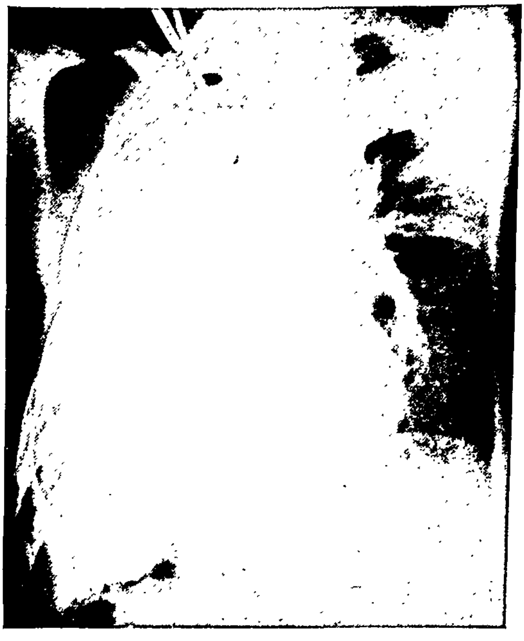

Fig. 1.—Case 1, F. O. Right pneumonectomy (tuberculosis). Angiogram taken three months after the operation. The pulmonary artery is displaced toward the operated side; its left branch is elongated.

We are reporting thirteen cases of pulmonary resection in which angiographic studies were made. The patients in our series were from 18 to 50 years of age, nine men and four women; three patients were pneumonectomized, two of them due to tuberculosis and one due to bronchiectasis with abscess formation; in the other ten cases lobectomy was performed (two lobes in four cases), four were bronchiogenic cysts infected, one inflammatory stenosis of the lower left bronchus, one carcinoma of the lower left bronchus in which a palliative lobectomy was performed as the bronchial obstruction produced the formation of a distal abscess, two bronchiectasis, one abscess, and one tuberculosis.

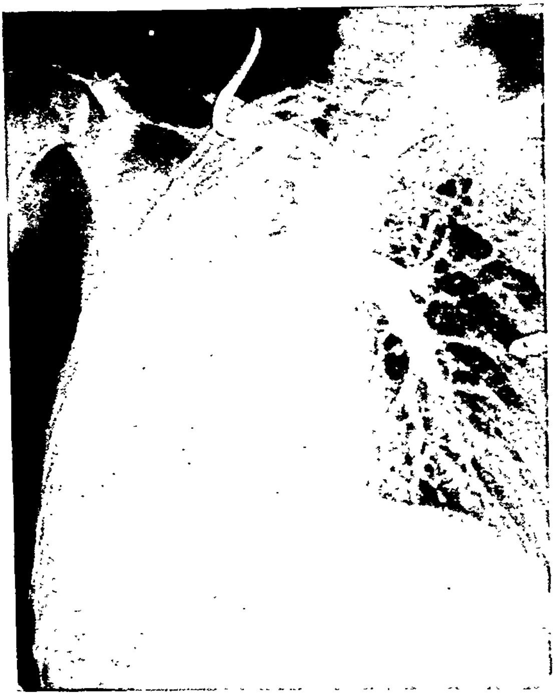

Fig. 2.—Case 2, D. H. Right pneumonectomy (bronchiectasis with abscess formation). Angiogram taken two years and four months after the operation. The displacement of the pulmonary artery is more marked than in the former case (Fig. 1); the pulmonary artery is longer and its branches are more separated.

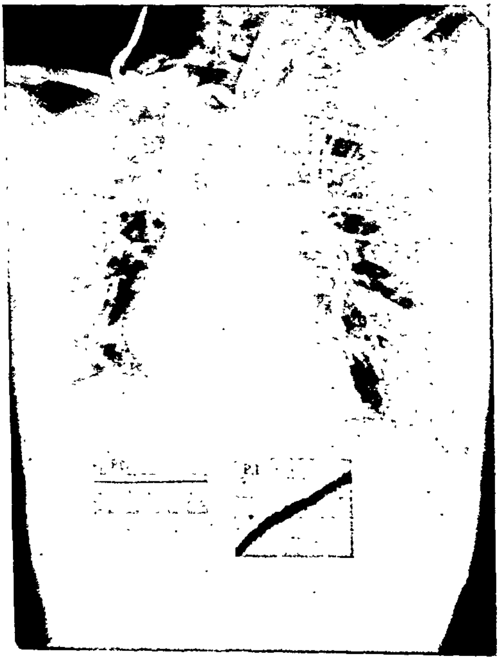

Fig. 3.—Case 12, M. V. Angiogram taken before the patient was operated. Notice the normal position of the mediastinum.

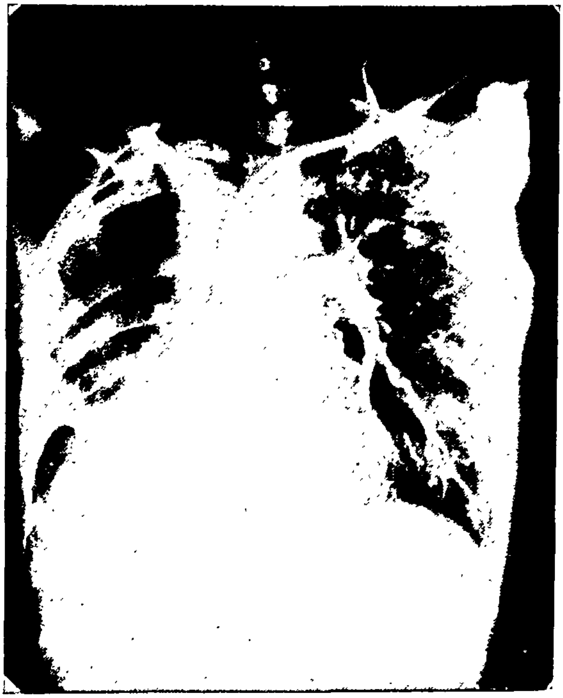

Fig. 4.—Case 12 after right pneumonectomy (pulmonary tuberculosis). Angiogram taken eighteen days after operation. The mediastinum is not displaced. The right branch of the pulmonary artery is sectioned.

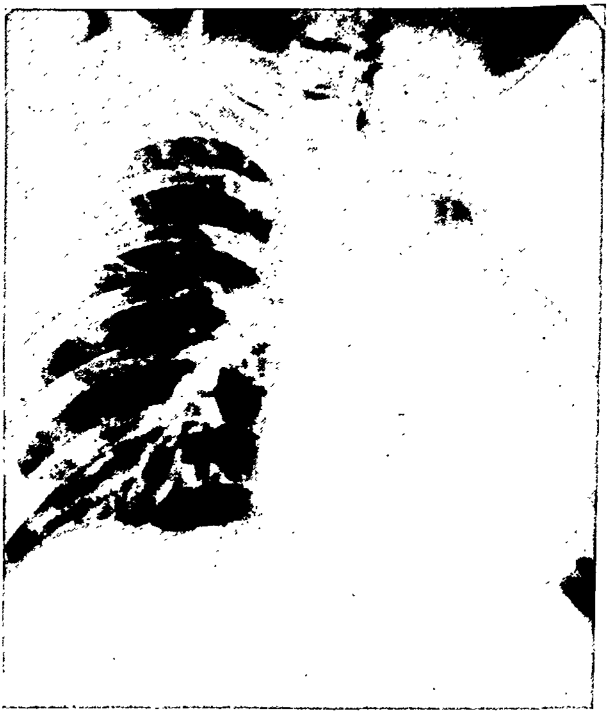

Fig. 5.—Case 3, R. M. Lobectomy of the upper left lobe (cyst). Angiogram taken one year and four months after the operation. The pulmonary artery is displaced toward the operated side, the blood supply is diminished. This patient developed a postoperative empyema.

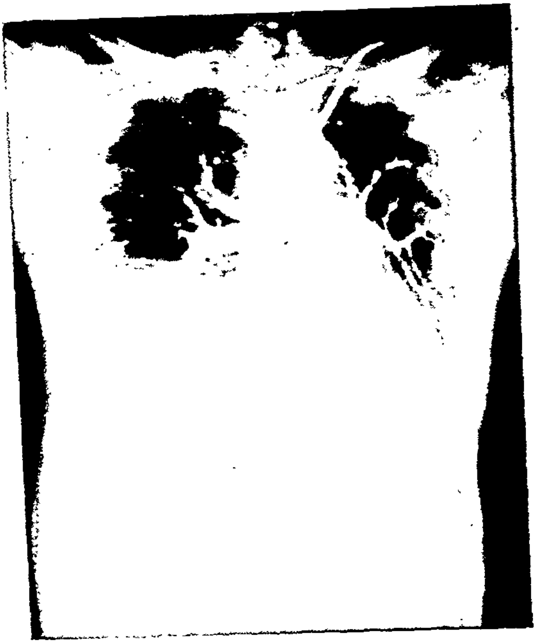

Fig. 6.—Case 8, C. G. Bilateral lobectomy (bronchiectasis). There is a diminished blood supply in both lungs.

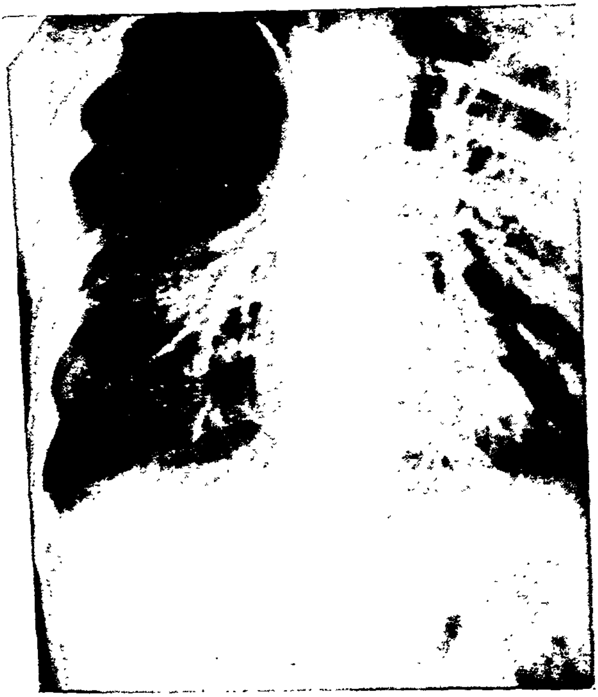

Fig. 7.—Case 7, Q. L. Lobectomy of the right upper lobe (cyst). Right pneumothorax. The blood supply to the rest of the pulmonary tissue appears to be normal.

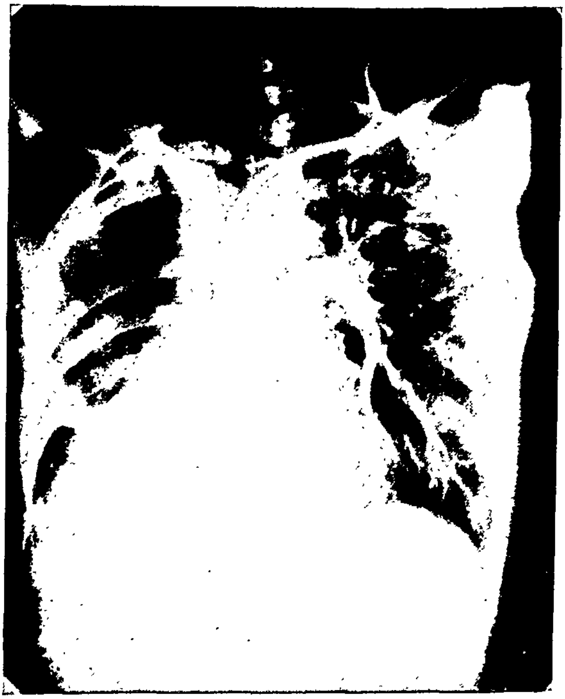

Fig. 4.—Case 12 after right pneumonectomy (pulmonary tuberculosis). Angiogram taken eighteen days after operation. The mediastinum is not displaced. The right branch of the pulmonary artery is sectioned.

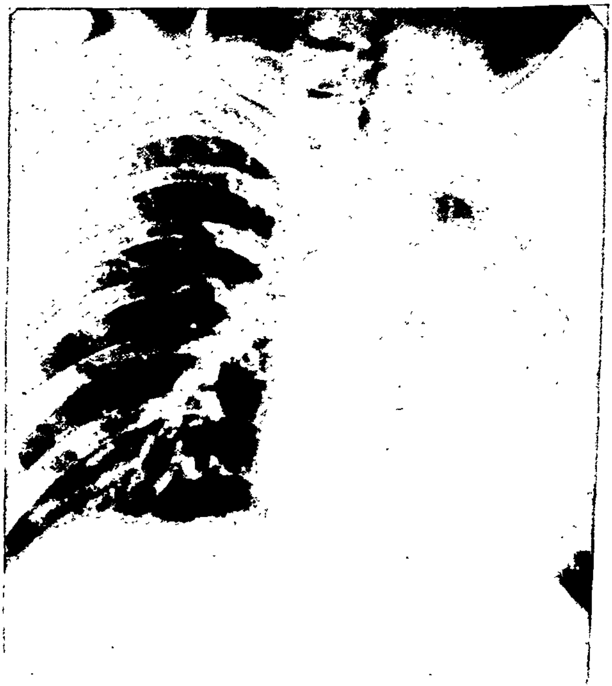

Fig. 5.—Case 3, R. M. Lobectomy of the upper left lobe (cyst). Angiogram taken one year and four months after the operation. The pulmonary artery is displaced toward the operated side, the blood supply is diminished. This patient developed a postoperative empyema.

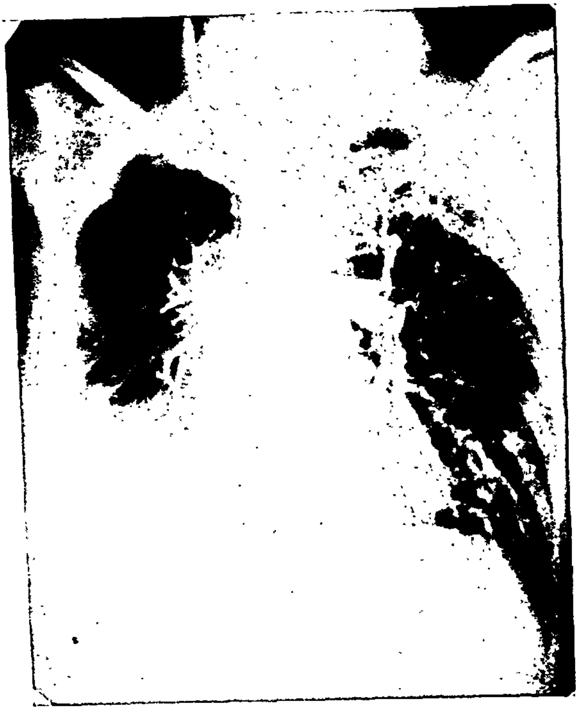

Fig. 9.—Case 11, R. B. Lobectomy of the lower and middle lobes (tuberculosis). Pulmonary hyperillumination, diminished circulation.

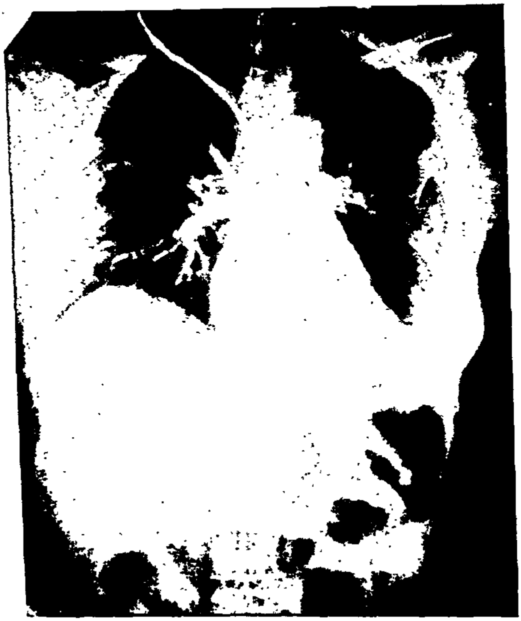

Fig. 10.—Case 13, A. A. Lobectomy of the left lower lobe (bronchiogenic carcinoma with distal abscess formation). Pulmonary hyperillumination, there is also a diminished circulation.

The cases studied are divided into two groups: (1) pneumonectomized patients, and (2) lobectomized patients. In the first group we report three cases. CASE 1.—F. O. (Fig. 1) on whom the operation was performed three months before the angiographic study. The pulmonary artery is displaced toward the operated side, its left branch is elongated, its smaller branches show diastasis, and the blood supply at the periphery is somewhat diminished. CASE 2.—D. H. (Fig. 2) in which angiographic studies were made two years and four months after pneumonectomy (right lung), the pulmonary artery suffered a marked displacement toward the operated side. There is a considerable dilatation, and its walls are elongated; its branches at the periphery of the lung pass through the mediastinum, and there is a separation between them. The third case (number 12 in Table I), M. V. (Figs. 3 and 4), was studied before pneumonectomy and eighteen days after the operation. There are no visible changes; the pulmonary artery is in its normal position, and its branches seem to be normal.

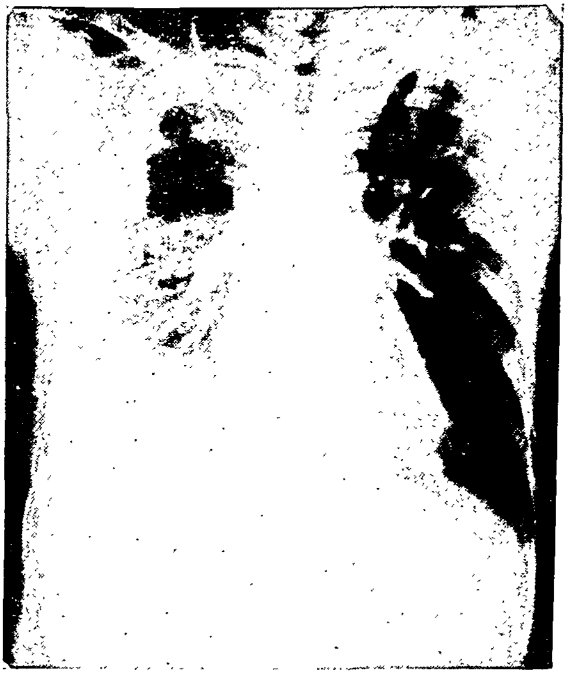

Fig. 8.—Case 9, M. S. Lobectomy of the right upper lobe (abscess). There is no alteration of the pulmonary artery as compared with the opposite side.

In the rest of the cases lobectomy was performed; CASE 3.—R. M. (Fig. 5), an 18-year-old patient on whom angiographic studies were made one year and four months after the operation, shows the pulmonary artery to be displaced toward the operated side, and a diminished blood supply to the rest of the lobes. It is interesting to note that this patient developed an empyema which was later treated by open drainage, and finally the total re-expansion of the other lobes was obtained. CASE 8.—C. G. (Fig. 6) had bilateral lobectomy and was studied four months after the first operation and twenty days after the second, the stem of the pulmonary artery is not displaced although there is a diminished blood supply at the bases. CASES 7 and 9.—Q. L. (Fig. 7) and M. S. (Fig. 8), on whom the rest of the lobes did not re-expand after the operation and, when the angiographic studies were made, they were seen to have pneumothorax. There is no displacement of the pulmonary artery, and its branches are not separated nor the blood supply diminished in the other lobes. In the rest of the cases there is a diminished blood supply to the lobe or lobes extirpated (Figs. 9 and 10).

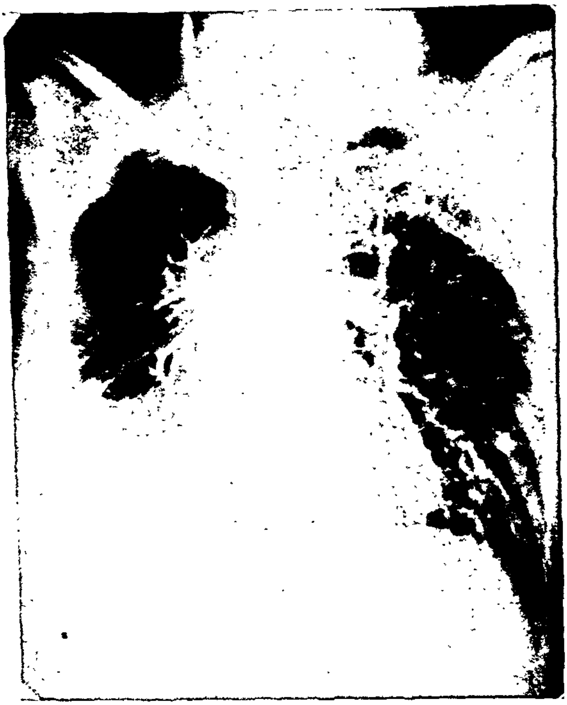

Fig. 9.—Case 11, R. B. Lobectomy of the lower and middle lobes (tuberculosis). Pulmonary hyperillumination, diminished circulation.

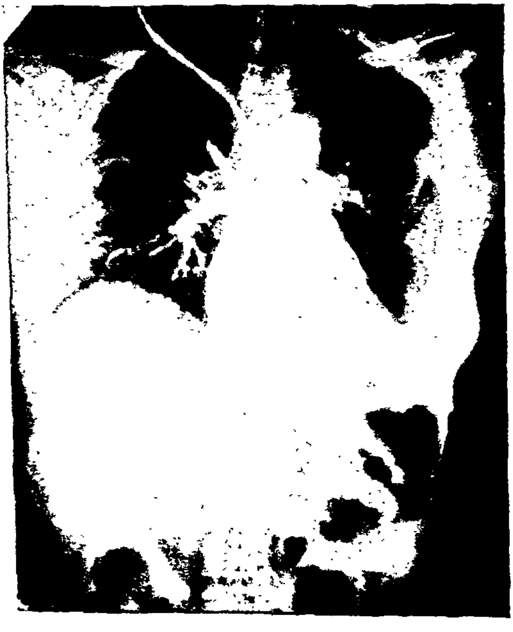

Fig. 10.—Case 13, A. A. Lobectomy of the left lower lobe (bronchiogenic carcinoma with distal abscess formation). Pulmonary hyperillumination, there is also a diminished circulation.

TABLE I

| NAME | AGE | SEX | DIAGNOSIS | TYPE OF OPERATION | DATE | DATE OF ANGIOGRAPHY |
|---|---|---|---|---|---|---|
| 1. F. O. | 25 | M | Right pulmonary tuberculosis | Pneumonectomy, right | Feb. 2, '46 | Jan. 1, '47 |
| 2. D. H. | 38 | F | Right bronchiectasis and abscess | Pneumonectomy, right | Jan. 29, '48 | June 13, '50 |
| 3. R. M. | 18 | M | Cyst, upper left lobe | Lobectomy, upper left | June 30, '49 | Oct. 13, '50 |
| 4. N. V. | 20 | M | Cyst, right upper lobe | Lobectomy, upper right | Nov. 15, '49 | May 26, '50 |
| 5. E. H. | 34 | F | Cysts, upper and middle lobes | Lobectomy, upper and middle | Feb. 9, '50 | May 30, '50 |
| 6. I. E. | 30 | M | Bronchiectasis, lower and middle lobes | Lobectomy, lower and middle | March 2, '50 | April 15, '50 |
| 7. Q. L. | 26 | M | Cyst, upper right lobe | Lobectomy, upper right | April 22, '50 | May 22, '50 |
| 8. C. G. | 45 | M | Bronchiectasis, lower and middle left | Lobectomy, lower and middle | July 10, '50 | Dec. 7, '50 |
| 9. M. S. | 35 | M | Abscess, upper right lobe | Lobectomy, upper right | Nov. 18, '50 | Aug. 17, '50 |
| 10. J. Z. | 18 | M | Inflammatory stenosis, lower left bronchus | Lobectomy, lower left | July 15, '50 | Oct. 31, '50 |
| 11. R. B. | 29 | F | Pulmonary tuberculosis, right lower and middle | Lobectomy, lower middle | Sept. 19, '50 | Dec. 13, '50 |
| 12. M. V. | 26 | M | Right pulmonary tuberculosis | Pneumonectomy, right | Nov. 27, '50 | Dec. 15, '50 |
| 13. A. A. | 50 | F | Carcinoma, lower left bronchus, also abscess | Lobectomy, lower left | Dec. 5, '50 | Dec. 20, '50 |

## DISCUSSION

In the cases we studied no method was used to fill the cavity remaining after pneumonectomy or lobectomy; instead the rest of the pulmonary tissue was left to re-expand and fill the space left after the operation. We observed that in all cases the lung tends to expand, thus representing more tissue surface to be supplied, as demonstrated by angiography, with the same number of blood vessels since these are unable to multiply themselves, giving rise to a respiratory insufficiency in the affected lung.

We observed also that both the pulmonary artery and mediastinum tend to be displaced toward the pneumonectomized side, and that such a displacement, also found in cases of postoperative complications, is more marked as time passes.

In the cases with pneumothorax there is no alteration of the pulmonary vessels, since the presence of the pneumothorax acts to prevent the overexpansion of the remaining pulmonary tissue.

## CONCLUSIONS

1. We use angiography as a method of studying the function of the lung.

2. With this method we are able to observe the alterations which pulmonary resection produces in the mediastinum.

3. The angiogram gives information as to the state of the mediastinum and pulmonary vessels when the patient has developed postoperative complications.

4. The mediastinal displacement is more marked the longer the time between pneumonectomy and the angiographic study.

5. Allowing the rest of the lobes or the other lung to re-expand and fill in the cavity left after lobectomy or pneumonectomy gives rise to a state of respiratory insufficiency.

6. The substitution of the lung tissue extirpated by a collapse method may be useful to prevent the total re-expansion of the rest of the pulmonary tissue.

7. We condemn pneumonectomy from a functional point of view, and advocate the extirpation of as little pulmonary tissue as the disease may allow in order to prevent respiratory insufficiency.

## SUMMARY

Thirteen cases of pulmonary resection (lobectomy or pneumonectomy) are *presented in which angiographic studies were made. After pneumonectomy,* the mediastinum is found to be displaced toward the operated side. Such a displacement is more marked the longer the time between the operation and the time in which angiography is performed. In lobectomized patients there is a diminished blood supply to the rest of the lobes as shown by the angiograms. In all cases there is an overdistention of the other lung after pneumonectomy, or other lobes after lobectomy.

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# SPONTANEOUS CONTRALATERAL PNEUMOTHORAX COMPLICATING THORACIC SURGICAL PROCEDURES

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THE general trend of reduced mortality and morbidity following thoracic surgical procedures during the past two decades has created a challenge for the further betterment of operative results. This report is being made to record additional cases and re-emphasize the potential danger of an uncommon serious postoperative complication which, if recognized and treated promptly, can favorably alter the course of the few patients in whom it may occur.

## REVIEW OF LITERATURE

A review of the surgical literature reveals relatively few references to spontaneous contralateral pneumothorax following thoracic operations. In 1936, Coryllos and Ornstein[3] reviewed the complications of the surgical treatment of bilateral pulmonary tuberculosis and discussed a situation which was similar in some respects to the complication which forms the basis for this report. In performing thoracoplasty on a group of patients with bilateral tuberculosis, an artificial pneumothorax was maintained on the opposite side. Twenty-two instances of superimposed collapse of a spontaneous nature occurred on the contralateral (pneumothorax) side in the postoperative period, four deaths resulting as a direct consequence of this complication. On the basis of thoracoscopic examinations, the etiologic mechanism was found to be an apparent rupture of an emphysematous bleb on the surface of the partially collapsed lung. This mechanism is taken up subsequently in the discussion of etiology. However, we are concerned at the present time with the incidence of spontaneous pneumothorax, with or without a tension factor, on the contralateral side or "good lung."

Stephens'[8] report is the earliest available description in detail of contralateral pneumothorax following intrathoracic operations. (See Table I and the review of reported cases.)

In his detailed review of 345 cases of bronchiogenic carcinoma, Bjork[2] analyzed the operative complications in 152 cases in which thoracotomy was performed. No instance of contralateral pneumothorax was recorded. Likewise, an earlier report by Ochsner and DeBakey[7] on surgical considerations of carcinoma of the lung failed to mention any encounter with this unusual post-

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TABLE I. REPORTED CASES

| CASE | OPERATION | POSTOPERATIVE OCCURRENCE OF PNEUMOTHORAX | OUTCOME | TENSION FACTOR |
|---|---|---|---|---|
| 1. Stephens—Case I | Exploratory thoracotomy | Operative day | Died | Present |
| 2. Stephens—Case II | Left pneumonectomy | Operative day | Died | Present |
| 3. Stephens—Case III | Lobectomy, right lower lobe | Operative day | Survived | Present |
| 4. Melick and Gutekunst | Left pneumonectomy | 60 days postoperative | Survived | Present |
| 5. Gleason and Kent | Lobectomy, left lower lobe | 3rd postoperative day | Survived | Absent |
| 6. Case I (this report) | Lobectomy, right upper lobe | 2nd postoperative day | Survived | Present |
| 7. Case II (this report) | Open thoracotomy drainage, right | 10th postoperative day | Died | Present |
| 8. Case III (this report) | Thoracoplasty, right, 4th (anterior) stage | 33rd postoperative day | Survived | Absent |

operative episode. Alexander's[1] text on the collapse therapy of pulmonary tuberculosis mentions the possibility of spontaneous pneumothorax occurring on the contralateral side during artificial pneumothorax treatment. He states that an immediately perilous condition may develop, and several cases are described in which this situation occurred. However, contralateral spontaneous pneumothorax is not referred to in the discussion of thoracoplasty complications.

More recent references in the literature are devoted mainly to the few case reports listed in Table I and described in some detail below.

The apparent infrequency of this complication may be due to several factors. The cause for sudden death of a patient in the early postoperative period may not be elucidated without autopsy examination; hence, failure to recognize the actual mechanism involved. Further, without ante-mortem suspicion or outright diagnosis, the pathologist's report may include the findings of "complete atelectasis" or "massive collapse" of the lung without specific effort being made to carefully scrutinize any small emphysematous blebs or the integrity and continuity of the mediastinal wall.

Table I lists the cases available for study and review including the three patients described in this report. A brief summary of each case follows:

Stephens' first case followed a left exploratory thoracotomy for inoperable carcinoma. Five hours postoperatively subcutaneous emphysema appeared, and ten hours later the wound was reopened with the demonstration of an open bronchus. Closed tube drainage was used, but the patient died twenty-five hours postoperatively and the diagnosis of a collapsed opposite lung was made at autopsy.

In the second case reported by Stephens, a left pneumonectomy was performed on a 40-year-old man for bronchogenic carcinoma. The patient died at the completion of the operation when turned over on his back. Autopsy showed the right lung entirely collapsed.

Stephens' third case occurred after a right lower lobectomy for bronchiectasis in a 10-year-old boy. When found that forced positive pressure by the anesthetist was necessary for completion of the operation, a needle was inserted into the left thorax and free air found. This patient survived.

It was assumed that the etiologic factor in each of these three cases of contralateral pneumothorax was a communication to the unoperated side through a probable tear of both layers of the mediastinal pleura. However, such a tear could not be demonstrated in the two cases which came to autopsy.

Melick and Gutekunst[6] reported a case in which a left pneumonectomy was done for tuberculosis complicated by bronchiectasis. Early in the postoperative course marked cardiac arrhythmia occurred which was eventually converted to normal. Sixty days following the operation sudden severe dyspnea developed. Apparently atelectasis was considered the most likely diagnosis, and bronchoscopy performed. However, no evidence of obstruction was found, and the diagnosis of contralateral tension pneumothorax was confirmed when the chest tap was done. Initial aspirations of air gave only temporary relief of dyspnea so that closed catheter suction was utilized and maintained for thirteen days. Thereafter, the lung remained re-expanded, and recovery was uneventful.

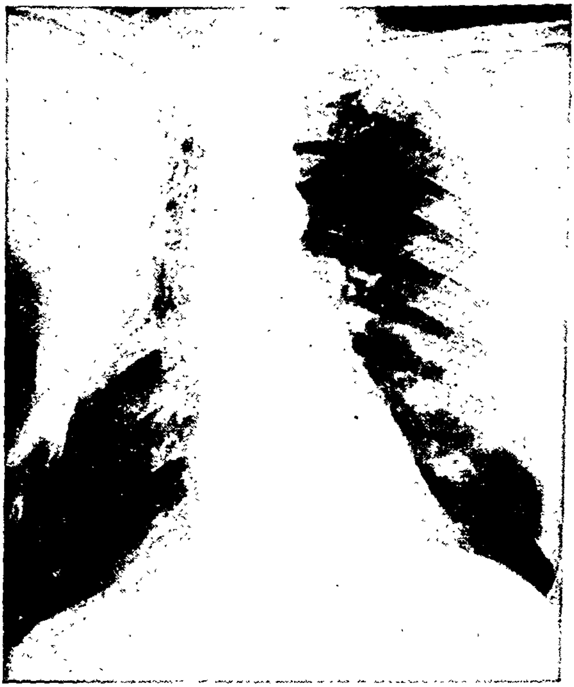

Fig. 1.—Case 1. Preoperative roentgenogram of the chest prior to right upper lobe lobectomy. The left lung presented no abnormal findings.

A case has been described by Gleason and Kent[4] in which a left lower lobe resection had been performed for bronchiectasis with the appearance of a right spontaneous pneumothorax on the third postoperative day. The patient manifested only moderate dyspnea without cyanosis. When conservative therapy was ineffective, the chest was opened nineteen days later and a leaking emphysematous bleb excised. Recovery followed. The authors stated that tension pneumothorax with mediastinal shift did not occur, but, had it been present, the respiratory embarrassment would undoubtedly have been much more marked.

## REPORT OF CASES

The three cases to be described in some detail occurred on the thoracic surgery service of the Veterans Administration Hospital, Wood, Wisconsin. Two of the cases were observed within a relatively short period of time, and our attention was directed to the dangerous potentialities of this operative complication.

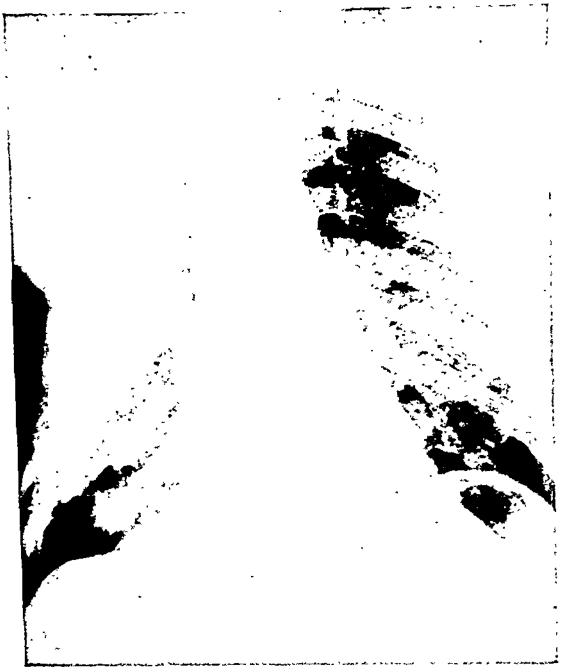

Fig. 2.—Case 1. First postoperative day film following right upper lobectomy showing satisfactory re-expansion on the right and a normal-appearing contralateral lung.

CASE 1.—R. F. (VA No. 125 216), a 53-year-old white man, had had a seven-rib right thoracoplasty for tuberculosis completed in 1947. His sputum remained negative until November, 1949, when it again became positive (Fig. 1). Resectional surgery was recommended but delayed because of a surprisingly low respiratory reserve. The ventilatory studies revealed the following:

| | | | |
|---|---|---|---|
| Reserve air | 644 | Expected MBC | 102.4 |
| Complementary air | 1577 | Per cent of expected | 50% |
| Vital capacity | 2033 | Walking ventilation | 13.22 |
| Maximum breathing capacity | 51.06 | W. V./MBC | 0.26 |

Bronchospirometry showed 25 per cent oxygen consumption on the right, 75 per cent on the left.

A right upper lobectomy was performed (W. W.) on Jan. 31, 1951, under general anesthesia with endotracheal intubation. The operation was completed without unusual event, but it was specifically noted that the mediastinal wall was fairly rigid and "frozen." The postoperative condition was satisfactory, and the portable bedside roentgenogram showed good expansion of the right middle and lower lobes and the left lung to be negative. The first postoperative day was likewise uneventful with two drainage tubes functioning well with connection to water sealed drainage bottles (Fig. 2). On the morning of the second postoperative day, the temperature was recorded as 98.6° F., pulse rate 120 per minute, and moderate dyspnea noted, with slight subcutaneous emphysema of the neck extending up to the left side of the face. At 4:00 P.M. in the afternoon the dyspnea became marked, cyanosis evident, and little relief afforded by an oxygen mask conveying 8 liters per minute. Cursory examination showed the drainage tubes on the right (operative side) to be open and functioning; the left chest was tympanitic with evidence of mediastinal shift to the right, the point of maximum cardiac impulse being palpated at the left border of the sternum. During the examination the patient's condition became progressively more critical with extreme dyspnea, leading to gasping, infrequent respiratory efforts, unconsciousness, and a weak almost imperceptible pulse. The portable x-ray machine was set up in the recovery room, but the actual film was not taken when the diagnosis was realized, with the fear that the few minutes spent adjusting the cassette and the machine would be disastrous. Immediate thoracentesis was carried out in the second left interspace anteriorly; free air under pressure was found in the pleural space. With negative suction on the needle, large volumes of air were aspirated, and a trocar was inserted just lateral to the needle allowing installation of a catheter connected to continuous suction with an intervening water seal bottle. Dramatic improvement followed, and within twenty minutes the patient recovered sufficiently to speak, remove his oxygen mask, and cough. Subsequent portable roentgenograms showed almost complete re-expansion of the left lung with the catheter in place (Fig. 3). The patient's condition remained critical, however, for the next few days with atelectasis developing in the right middle and lower lobes necessitating daily bronchoscopic aspiration. After progressive clearing of the right side, continued expansion of the left side, and loss of the subcutaneous emphysema, the tubes were removed from the right side on the fourth postoperative day and the catheter from the left side on the eighth postoperative day. Three hours after the

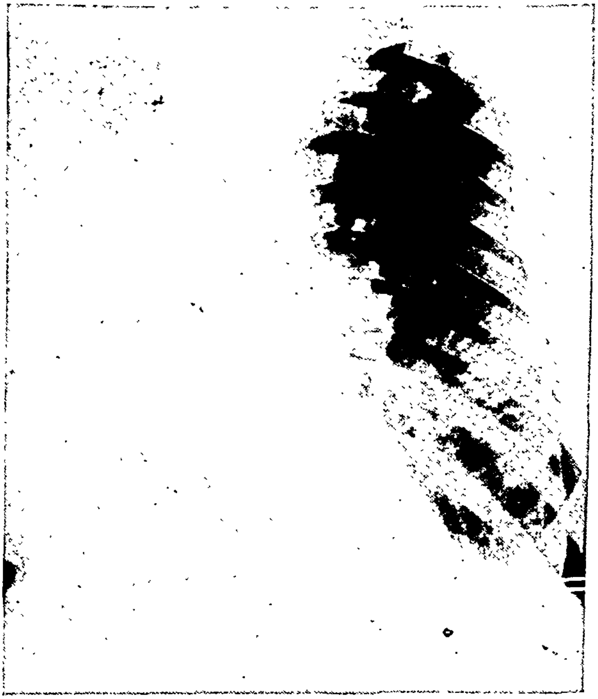

Fig. 3.—Case 1. Roentgenogram taken on the 6th postoperative day, four days after the spontaneous contralateral pneumothorax. The catheter can be visualized in the left pleural space. Note the right lower lobe atelectasis requiring bronchoscopic aspiration.

catheter from the left pleural space had been removed, signs and symptoms of recurrence of the left tension pneumothorax and mediastinal shift became apparent. Being prepared for this possibility a roentgenogram was obtained and a catheter reinserted promptly (Fig. 4). Following this episode, the catheter suction was maintained for twelve days, and, following removal, the left lung remained fully expanded with progressive recovery (Fig. 5).

Gross examination of the surgically removed lobe revealed a tuberculous cavity 1.5 cm. in diameter. Over the apical portion multiple small bullae and blebs measuring 0.5 cm. in diameter were found. Micro sections revealed tuberculosis, and, peripherally, marked emphysematous changes with dilatation of the alveoli and rupture of the alveolar sacs.

Fig. 4.—Case 1. Recurrence of the pneumothorax on the left following removal of the intrapleural catheter on the eighth postoperative day.

CASE 2.—G. K. (VA No. 126 540), a 33-year-old white man, was admitted to the thoracic surgical service with a right empyema treated at another hospital by open thoracotomy drainage. A bronchopleural fistula had developed, and the right lung was but 50 per cent expanded. The patient was chronically ill, and his subsequent course was a long and difficult one. On Dec. 26, 1950, a decortication of the right lung and a right middle lobectomy (W. W.) were performed. A stormy postoperative course followed with retraction of the lung and accumulation of pleural fluid (Fig. 6). Open thoracotomy became essential, and this was done on Feb. 13, 1951. A large Fryer type rubber tube was used to maintain the open drainage and the postoperative course was satisfactory, with progressive improvement in his general condition. On the tenth postoperative day at 4:30 P.M. he noted persistent mild dyspnea and, following his evening meal, the dyspnea suddenly became severe. When seen by the resident physician he was sitting up in bed, breathing with extreme difficulty; no cyanosis was evident, the pulse rate was 120 per minute, and regular. Examination of the left chest revealed some flatness to percussion over the left paravertebral region, distant breath sounds on auscultation with no râles or other adventitious sounds, and evident use of the accessory muscles of respiration. Oxygen therapy was begun as soon as the patient was seen, and a portable bedside chest roentgenogram was taken. Dyspnea increased and cyanosis appeared.

With a tentative diagnosis of tracheal or bronchial obstruction, an attempt was made to pass an endotracheal tube but was unsuccessful due to the onset of convulsive movements with muscle rigidity. A tracheotomy was performed immediately, endotracheal suction being instituted along with artificial respiration. He failed to respond to these measures, and died approximately fifteen minutes after the onset of the acute symptoms. The roentgenogram mentioned above (Fig. 7) was not developed in time to be read before the patient's demise.

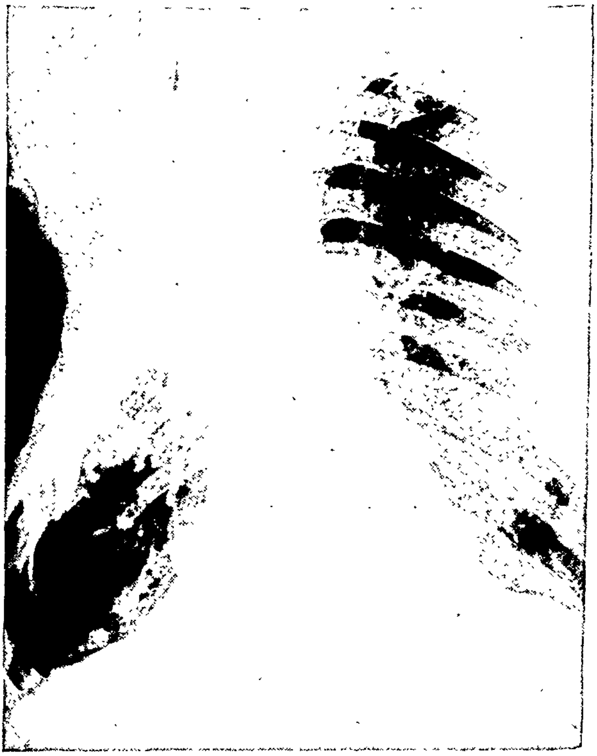

Fig. 5.—Case 1. Full re-expansion of the left lung after a total of eighteen days' intrapleural catheter suction.

Autopsy examination was preceded by the usual method of demonstrating tension pneumothorax: an incision over the left second interspace anteriorly with elevation of the wound edges produced an excavation which was filled with water. Incision through the intercostal muscles and pleura allowed sudden outward gushing release of the air under tension. The left lung was found completely collapsed. Over the apex of the left upper lobe, numerous small emphysematous bullae were present, one of which, measuring 1.5 cm. in diameter, was found to be ruptured. The site of rupture was immediately adjacent to the attachment of a narrow adhesive band, the opposite end of the band being fixed to the parietal pleural surface in the apical area. No tear of the parietal pleura could be found. After removal of the left lung, re-expansion under water demonstrated the exit of air from the ruptured site.

Case 3.—E. M. (VA No. 105 474), a 29-year-old white man, who had a diagnosis of pulmonary tuberculosis made in 1943. As a patient in a U. S. Naval Hospital, artificial pneumothorax on the left was instituted in March, 1943, and maintained until February, 1948. Pneumonolysis was carried out on the left in May, 1943, and again in July, 1943. The sputum and gastrics had become negative following collapse therapy and remained negative until

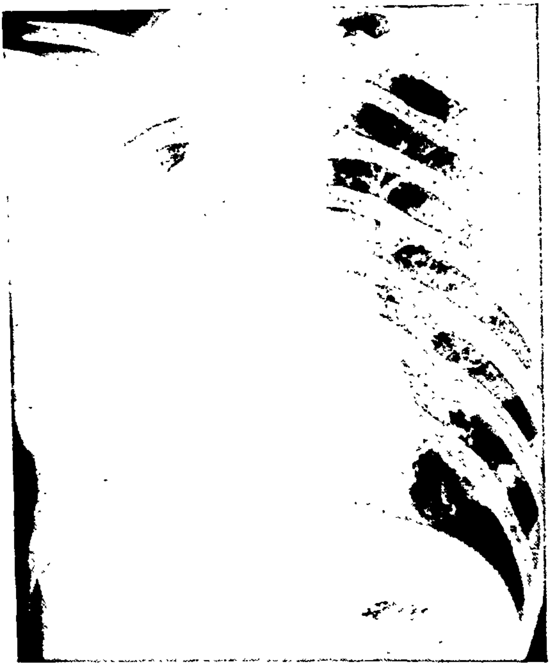

Fig. 6.—Case 2. Chest roentgenogram prior to open thoracotomy drainage on the right. The left lung appeared normal.

Fig. 7.—Case 2. Portable bedside chest roentgenogram ½ hour ante mortem, revealing the marked collapse of the left lung.

February, 1948. The patient was admitted to Wood Veterans Administration Hospital in June, 1948, at which time the left artificial pneumothorax was discontinued. Re-expansion was satisfactory, and subsequent roentgenograms revealed fibrocaseous tuberculosis involving the right upper lung, with several areas suggestive of cavitation. A course of streptomycin was given and right thoracoplasty recommended (Fig. 8). A six-rib right thoracoplasty was performed in stages, being uneventful, up to his ninth postoperative day when he complained of dyspnea. Fluoroscopy revealed a left pneumothorax which responded favorably to one aspiration and the lung was completely re-expanded. On the thirty-third postoperative day following his last stage, the pneumothorax recurred (Fig. 9) and, although symptoms were minimal, it required three successive aspirations for re-expansion.

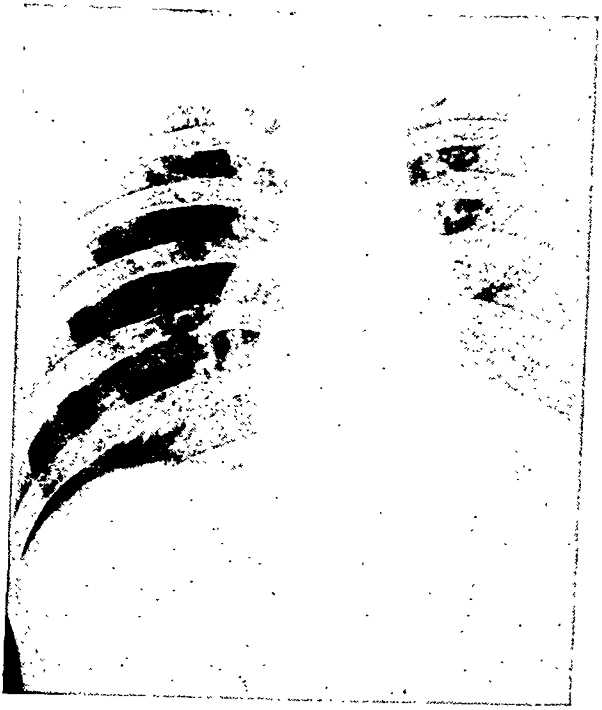

Fig. 8.—Case 3. Roentgenogram of the chest taken prior to the performance of thoracoplasty on the right side. The left lung showed some pleural thickening.

## DISCUSSION

With regard to the etiologic factors involved in the production of a spontaneous contralateral pneumothorax in the postoperative period, several mechanisms have been proposed. Stephens[8] maintained that each of his three cases was due to communication through the mediastinal wall, although the site could not be demonstrated in the two cases which were examined post mortem. Knoepp,[5] in discussing the importance of various mechanical and circulatory postoperative pulmonary complications, devoted some attention to spontaneous pneumothorax, but the only reference to contralateral tension pneumothorax following thoracic surgical procedures was a direct notation of Stephens' work. He also believed the mechanism was undoubtedly due to tearing the mediastinal pleura during the operative dissection. Melick and Gutekunst[6] did not suggest what the causative factor might have been in their cases. On the other hand, a ruptured emphysematous bleb was proved to be responsible in Gleason and Kent's[4] report. We have considered this latter mechanism to be the most feasible in each of the cases reported herewith.

In Case 1, several possibilities had to be considered: (1) an air leak at the lobectomy site with communication through both layers of mediastinal pleura; (2) a pleural tuberculous lesion on the contralateral side; and (3) a ruptured emphysematous bleb with or without an adjacent adhesive band and simultaneous parietal pleural tear. After consideration of the various aspects of the case and review of the operative and pathologic findings, the third mechanism seems most appropriate. In Case 2, this mode of production of the pneumothorax was adequately demonstrated at autopsy as described above. Acceptable evidence of the mechanism involved in Case 3 was lacking for a long time until the patient returned to the surgical service for collapse therapy for disease in the left apex. During the course of preliminary intrapleural poudrage and subsequent extrapleural lucite ball plombage, several large blebs were visualized in the apical area, the presence of which allowed circumstantial although indirect proof of the etiology of his previous spontaneous pneumothorax. Hence, on the basis of our experience with these few cases and the additional cases available for study, we believe that the ruptured emphysematous bleb assumes a predominant role in the etiology of this serious postoperative complication.

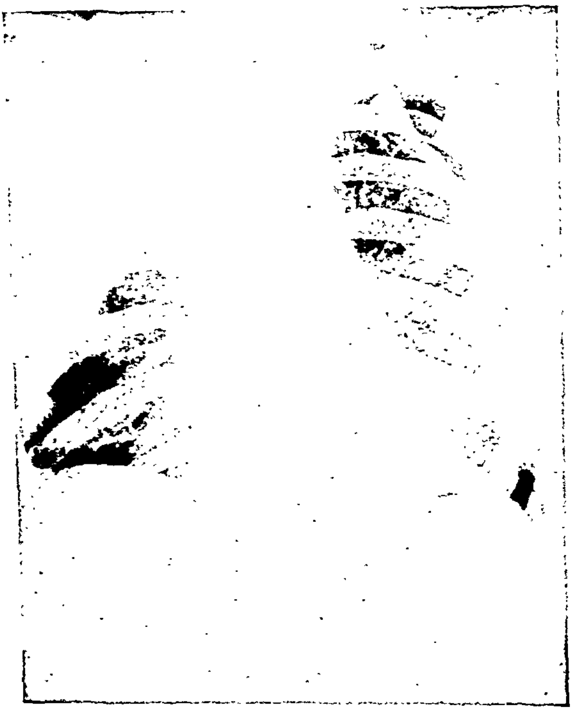

Fig. 9.—Case 3. Spontaneous contralateral pneumothorax on the left occurring thirty-three days following the last stage of thoracoplasty on the right.

Alexander,[1] in discussing perforation of the lung, has stated that any diagnostic measure which is not at the same time capable of relieving the symptoms of respiratory embarrassment is contraindicated. This statement implies that clinical indications must suffice to warrant the confirmatory diagnostic and likewise therapeutic application of immediate thoracentesis. Roentgenograms or fluoroscopy permit the loss of valuable time, and occasionally a patient's life. The validity of Alexander's statement is well demonstrated in Cases 1 and 2. In the first case the portable x-ray machine was actually being adjusted in the recovery room when the danger of the "time lost" factor was realized and definitive treatment carried out on the basis of the physical findings. In the second case, where death supervened fifteen minutes after onset of severe symptoms, the clinical picture was thought to be due to an obstructed airway and the patient treated accordingly. Time was taken to obtain a bedside roentgenogram of the chest, but the patient expired even before the film could be developed.

Treatment is simple and effective. Decompression of the pleural space by immediate thoracentesis with any large needle allows release of the high intrapleural pressure, correction of the mediastinal shift, and prompt relief from the severe subjective symptoms. Subsequent placement of an intrapleural catheter provides a satisfactory method of evacuating the air and connection to a continuous suction device. Thereafter, the management of the situation corresponds to that of any ordinary valvular pneumothorax.

## SUMMARY

1. Three cases of contralateral spontaneous pneumothorax following thoracic surgical procedures are described. The few instances of this operative complication previously reported are also reviewed.
2. Additional basis for the concept of the ruptured emphysematous bleb as an etiologic factor is recorded.
3. The awareness of the possibility of this serious complication by the thoracic surgeon and the resident physician is necessary to avoid mortality.

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# INTRATHORACIC MENINGOCELE: EXCISION, WITH THREE AND ONE-HALF YEARS FOLLOW-UP

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WITH the ever-increasing use of mass roentgenogram surveys of the chest, appreciable numbers of asymptomatic tumors are being detected. A not uncommon diagnosis is neurogenic tumor of the posterior mediastinum for which excision is usually recommended.

The following case, which was diagnosed as a neurofibroma of the posterior mediastinum, turned out to be an intrathoracic meningocele. The patient has been followed for three and one-half years after operation.

## CASE REPORT

E. F., a 31-year-old white woman, following a chest survey which had been done at the cotton mill where she worked, was told to see her doctor, since a tumor was seen on a roentgenogram.

On close questioning, the only symptom admitted was "occasional stiffness of the neck" which was mild. In addition she stated that she had had two roentgenograms of her chest previously in Army surveys while she was employed as a civilian helper in an army air depot, once in 1944 and once in 1945 (two and three years previously). She had never been notified that anything abnormal had been found. Those roentgenogram or reports could not be obtained.

*Past History.*—Childhood: measles and mumps. Ever since birth small skin growths had been present almost all over the body. One maternal aunt and one paternal aunt had had similar lesions all their lives. The patient had no children. In 1939 appendix and "tumor of ovaries" were removed.

*Examination.*—(July 2, 1947.) The skin exhibited a typical picture of disseminated neurofibromatosis (Von Recklinghausen's disease). *Head:* The eyes were examined with particular reference to any degree of Horner's syndrome, but were entirely negative. Nothing else of note. *Neck:* Slightly deviated to patient's right. *Chest:* Slight dullness at the left apex with somewhat diminished breath sounds in this region. Heart was normal, blood pressure 110/70, left arm: 105/65, right arm. Vital capacity was 3.8 liters. *Abdomen:* Liver and spleen were not palpable. No masses or tenderness were present. *Extremities:* No neurologic findings of any kind in upper or lower extremities were found. There was no clubbing. *Laboratory:* Blood and urine examinations were normal. *Roentgenogram:* See Figs. 1 and 2.

Homogeneous density was present at the apex of the left chest, merging with mediastinum and revealing a sharply defined outer border. The lesion extended to the level of the sixth interspace posteriorly. Lateral films showed it to fill the whole apical region. Films for better bone definition (see Figs. 3 and 4) showed angular scoliosis with bony deformities of the seventh cervical and first thoracic vertebrae. The left halves of the seventh cervical and first thoracic vertebrae appeared to be narrowed or compressed. In retrospect, this area probably represents bone defect and/or widening of the foramina.

*Fluoroscopy:* Large dense mass was present filling the whole left apical region. Lower outline was sharp, with no pulsation. There was no change in outline on cough or straining. No shift of mediastinum on forced expiration was noted. The diaphragm moved well.

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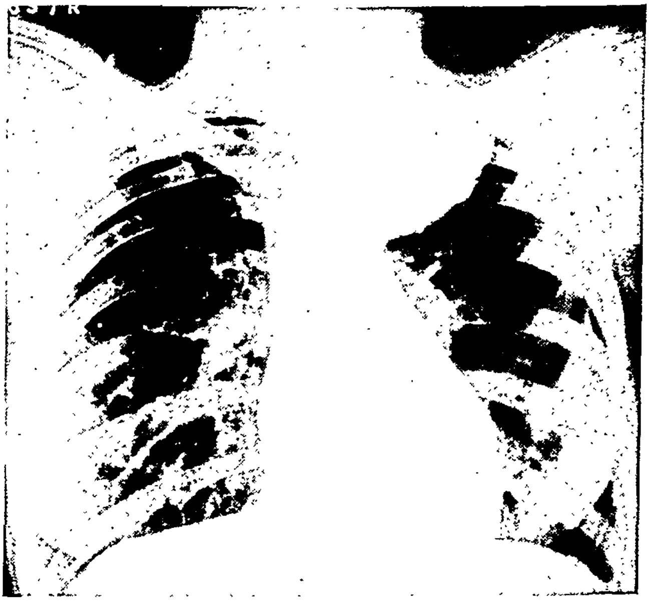

Fig. 1.—Standard chest plate showing large density at left apex with sharply demarcated curved inferior border.

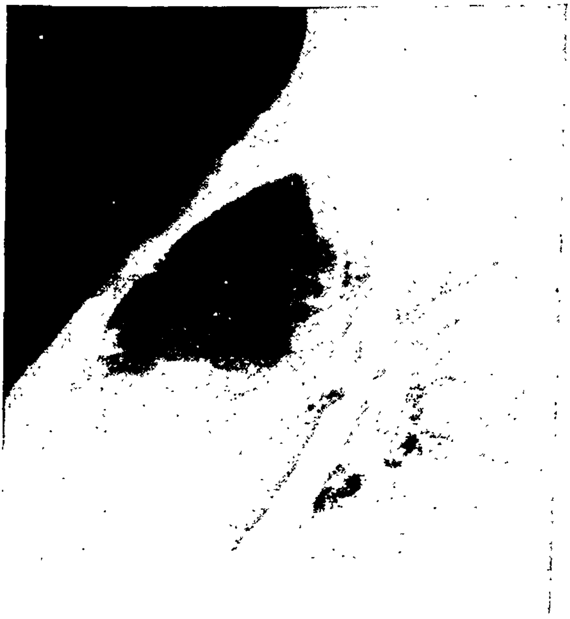

Fig. 2.—Lateral exposure with barium in esophagus. Lesion occupies whole apical region.

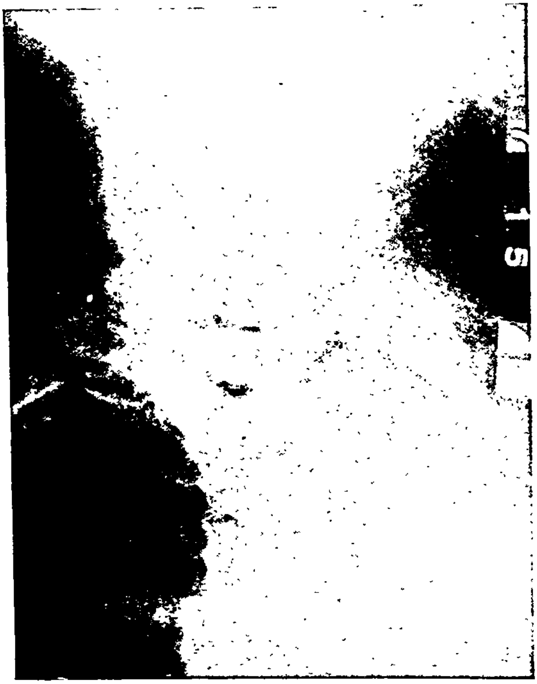

Fig. 3.

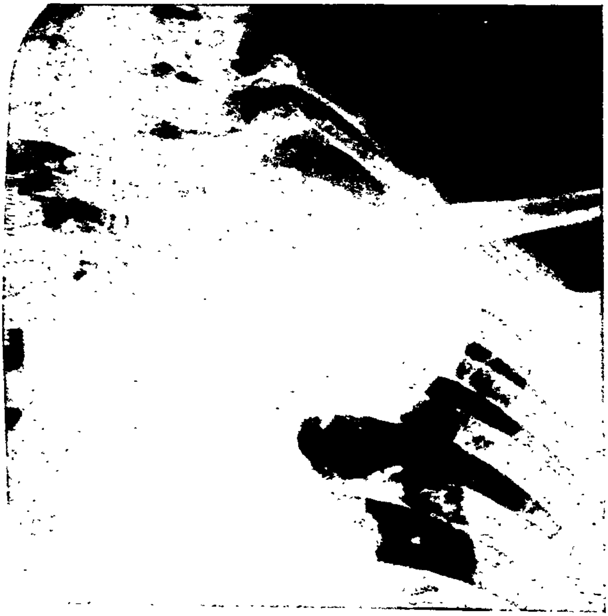

Fig. 4.

Figs. 3 and 4.—Bony deformities: bodies of seventh cervical and first thoracic vertebrae, compressed or collapsed in left lateral half, slight angular scoliosis, narrowing of posterior segment of second rib.

*Preoperative Diagnosis.*—(1) Multiple neurofibromatosis with large neurofibroma of posterior mediastinum (possible dumbbell type). (2) Bony deformities of the seventh cervical and first thoracic vertebrae, secondary to (1).

Operation was performed on July 16, 1947, at the Utica Memorial Hospital.

*Description of Operation.*—Under endotracheal cyclopropane anesthesia a high left posterolateral transpleural approach was used.

The fifth rib was removed subperiosteally from its angle to about the anterior axillary line. The pleura was entered through the bed of the resected fifth rib. The tumor appeared to occupy the whole apical region, and the lung was adherent to the entire lower aspect of the tumor. By combined blunt and sharp dissection, the lung was peeled away from the mass. This was done extrapleurally, as the lung was adherent to the parietal pleura overlying the mass.

During the liberation of the mass, it was noted that it was soft and fluctuant. Ten cubic centimeters of crystal-clear fluid were freely aspirated and, since it was suspected that this might be spinal fluid, it was saved for examination. It was decided to aspirate more of the fluid so as partially to collapse the sac and give better access to the pedicle.

About 120 c.c. of crystal-clear fluid were slowly withdrawn with no untoward reaction.

With the sac partially collapsed, the pedicle was easily approach, and could be felt to be coming from a bone defect in the anterolateral aspect of the vertebral column in the region of what we judged to be the first and second thoracic vertebrae. Two fingers could be placed just within the defect, the long axis being parallel to the long axis of the vertebral column. A double ligature of No. 1 chromic catgut was placed around the pedicle and drawn down tight at the base where it came out of the defect of the vertebral column.

The sac was cut off, leaving a liberal central stump to use in repair. Inspection revealed a slight leak of fluid just proximal to the ligature where there seemed to be a small tear. This was carefully repaired with No. 00 chromic catgut on an atraumatic needle. The defect was strengthened by suturing some pieces of parietal pleura over it after several sutures had been taken from the stump of the ligated sac to the near-by periosteum. Finally, strips of oxycel were laid over the whole area. Usual layer closure was done.

*Comment:* The sac contained at least 200 c.c. of crystal-clear fluid, and it was tough and resistant though thin.

*Postoperative Course.*—The patient did very well She was watched closely for any signs of leakage of spinal fluid, but at no time did she complain of headache or other unusual symptoms As a precaution she was kept in a lying position for the first twenty-four hours and then raised to a semisitting position She was ambulant on the third postoperative day.

A Horner's syndrome was noted postoperatively. Within about one week the temperature changes had become mild but the eye findings remained.

Analysis of the specimen of fluid removed from the sac was as follows:

| | |
|---|---|
| Specific gravity | 1.004 |
| Sugar | 96 |
| Chlorides | 720 |
| Total proteins | 43.6 |
| Cells | None |

One of the fleshy skin tumors removed from the back at the end of the operation showed it to be a neurofibroma.

The microscopic examination of the sac was compatible with that of dura with some nerve fibers attached on the outer aspect.

*Final Diagnosis.*—(1) Intrathoracic meningocele; (2) congenital bony defects of bodies and foramina of the seventh cervical and first thoracic vertebrae, with herniation of dural sac; (3) multiple neurofibromatosis.

*Follow-Up.*—The patient was last examined on Mar. 14, 1951. She presented no complaints. A Horner's syndrome of moderate degree was still present. The last roentgenogram (see Fig. 5) showed no evidence of recurrence.

## PREVIOUS REPORTS

At the time of operation, July 2, 1947, we had never heard of such a finding in the chest, and postoperatively we were quite uneasy as to whether excision of the sac had been the correct procedure or whether it would have been wiser to have backed out without disturbing it after the nature of the lesion was apparent.

At that time the only reports of similarly operated cases which we could obtain were those of Pohl,[1] in 1933, of which we were able to get an abstract in English, and of Ameuille and associates.[2] It is of interest that Pohl's patient also had neurofibromatosis, and that the preoperative diagnosis was intrathoracic neurofibroma. The chest lesion had been detected on an x-ray examination taken because of persistent cough following a mild attack of grippe. The patient, a woman, was operated upon, the sac excised, and a strip of muscle was used to cover the defect in the bone and the exposed spinal cord. However, postoperatively she developed an empyema which caused her death.

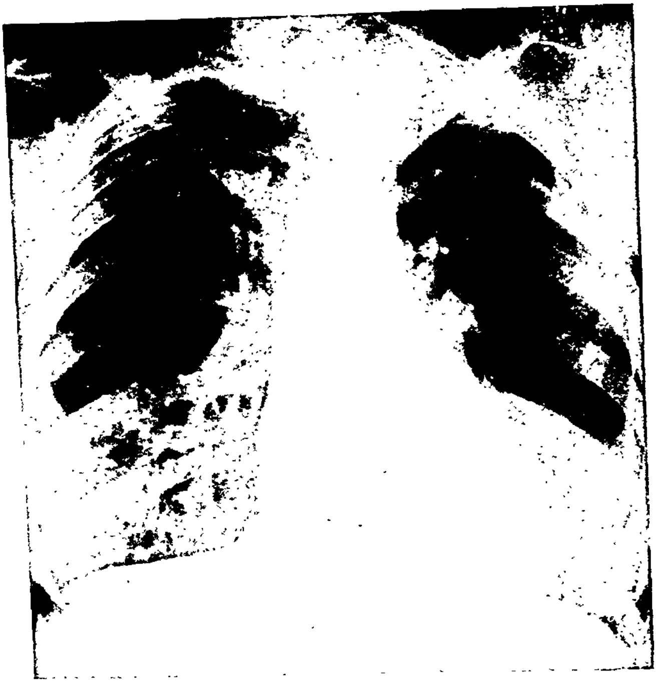

Fig. 5.—Last film over three and one-half years after surgery, showing no evidence of recurrence.

The case described by Ameuille, Wilmoth, and Kudelski[2] is of marked interest since the patient had had an x-ray examination eleven years previously, preoperative thoracoscopy was done, and a detailed autopsy description of the lesion is also given. Their patient was a 48-year-old woman, who for five years had complained of headaches, mild malaise, and weakness, and was considered a psychoneurotic.

In 1929 Duval had done an Albee graft because of lumbodorsal pain associated with kyphoscoliosis. X-ray study on her recent admission (1940) revealed a rounded shadow at the right base with well-defined borders, about 8 cm. in diameter. The lateral view placed it posteriorly and showed a rounded area of rarefaction in the body of the ninth thoracic vertebra. Artificial pneumothorax was done, and a thoracoscopic examination revealed what was thought to be a hydatid cyst.

Operation was performed, using a transpleural posterolateral route. The sac was easily detached from the right side of the spine, and at that time a punched-out opening was noted on the lateral aspect of the eighth and ninth thoracic vertebrae. "As it is not possible to close the openings in the vertebrae with the pleura, one simply dries the right hemithorax; the parietal pleura is sutured hermetically with all the parietal layers." The patient died on the eighth day with signs of meningitis and pleural exudate infected with *Staphylococcus aureus.*

The authors felt, in retrospect, that if the preoperative diagnosis had been made they could have emptied the sac and then used it, at least temporarily, to close the vertebral openings and so avoid the leakage of spinal fluid which they felt led to the death of their patient.

The next surgical cases that we became acquainted with were three which were presented in a paper by Byron and co-workers[3] at the meeting of the American Association for Thoracic Surgery in May, 1948. Several discussers also presented cases. The published paper will be referred to later.

Shortly after this, we found the report of Welch, Ettinger, and Hecht.[4] They reviewed the literature and presented a successful operative case of their own. In their search, only three reported cases of intrathoracic meningocele were found, two of them being the cases of Pohl and Ameuille, previously mentioned above. These writers also pointed out that their own, and two of the three reported cases, occurred in individuals with neurofibromatosis. At operation they excised the sac at the neck, closed the dura with interrupted silk, and covered the defect with a pedicle graft of the intrinsic dorsal musculature which was sutured to the edges of the intervertebral foramen. Their patient was a woman aged 41 years.

The next reported surgical case was that of Mendelsohn and Kay.[5] Once more the preoperative diagnosis was neurofibroma, in a woman patient with generalized neurofibromatosis. At operation the sac was opened widely, and the defect in the subarachnoid space was closed by imbricating the cyst wall and the parietal pleura with interrupted silk sutures. A small amount of talc was sprinkled over the pleura in this area to promote fibrosis. Roentgenograms "nine days and one month after the operation showed a complete recurrence. X-ray pictures one year later showed a complete regression, and the patient continues to be well except for low back pain with sciatic radiation suggestive of herniated vertebral disc." The patient refused further study.

Mendelsohn and Kay also pointed out the association with neurofibromatosis, and noted the high incidence in women. They referred to a personal communication with S. O. Freedlander, in which the latter stated that he had operated on a middle-aged woman with cutaneous neurofibromatosis who had a meningocele projecting into the posterior mediastinum. Kay observed an intrathoracic meningocele as an incidental finding at the autopsy table, but no mention was made of sex or the presence of neurofibromatosis.

The last published report is the complete paper by Byron, Alling, and Samson[3] which, as stated above, had been read in May, 1948, at the meeting of the American Association for Thoracic Surgery. They review the literature thoroughly, and the reader is referred to their article for the bibliography and further details of previously reported cases with the exception of those of Welch and associates, and Mendelsohn and Kay. Byron and co-workers operated on three patients, again with a preoperative diagnosis of neurofibroma of the mediastinum. One male patient had neurofibromatosis with *café au lait* spots, and another patient had *café au lait* spots but no neurofibromatosis. Bony changes of the vertebral column and ribs were present in two of the individuals.

Their first patient was treated by utilizing a portion of the cyst wall or dura as a flap to close the defect. This was then covered by a free pleural graft and a layer of Gelfoam. Postoperatively the patient experienced mild intermittent headache, but did well, and continued so after discharge. In their second patient no effort was made to treat the sac found at operation. The diagnosis of meningocele was confirmed by injection of Pantopaque into the sac and then visualizing it in the spinal canal. In their third case, a 61-year-old man, the cyst extended high into the neck, being embedded in the lower roots of the brachial plexus, making the exposure very difficult and resulting in considerable bleeding. Cardiac arrest occurred with no response to massage, etc.

In their review of previous cases, they mention the patient of Pohl, who died of empyema and meningitis; the patient of Ameuille and associates, who also died postoperatively of empyema and meningitis; a case reported by Schuller and Uiberall[6] diagnosed by myelography.

In the discussion of the paper of Byron and associates, Jerome Head reported an additional case in a 43-year-old man on whom a routine film of the chest revealed a globular tumor adjacent to the spine. Simultaneous puncture of the tumor and spinal canal revealed that withdrawing fluid from the tumor lowered the pressure in the spinal canal, and withdrawing fluid from the spinal canal lowered the pressure in the tumor. Examination of the specimens showed them to be comparable in appearance, cell count, and chemical analyses. Diagnosis of intrathoracic meningocele was made, and the patient was not operated upon.

## DISCUSSION

*Surgical Indications.*—From the surgical viewpoint the important question is—if this lesion is diagnosed preoperatively or encountered at thoracotomy, should it be attacked surgically or left alone?

Welch and collaborators apparently feel that surgery is warranted, but Mendelsohn and Kay state, "We are in doubt as to the advisability of operating on this type of lesion unless it shows progressive enlargement or causes symptoms. The defect is difficult to repair satisfactorily and might be associated with a high incidence of recurrence. It is felt that this is an innocuous tumor which is difficult to cure surgically, and that surgical interference is not indicated if the patient is asymptomatic.''

Byron and associates were of the opinion that ''in patients with relatively small sacs, operation is probably not justified unless serial chest roentgenograms reveal progressive enlargement to the point of progressive respiratory embarrassment.'' In the discussion of his paper Byron stated, ''I think there is some importance in making a preoperative diagnosis, because I do not believe operation is indicated in all cases. In the first place with a large bony defect, it is questionable in many cases whether the defect can ever be satisfactorily closed, and, second, since I believe that the meningocele is a herniation of the dura, I see no reason why herniation cannot recur.''

To the writer it seems reasonable enough to assume that surgery is indicated in a patient having symptoms. It also appears logical to defer surgery and follow the patient roentgenologically if the diagnosis is made preoperatively and the patient is asymptomatic. If the sac shows progression it would seem advisable to operate early to avoid technical difficulties which are greater in the large-sized sacs with marked bony erosion.

However, if the lesion is undiagnosed and encountered at thoracotomy, we feel that it should be treated surgically and not left alone. This attitude appears justified because a study of the reported cases and our own points to the fact that this is a *progressive* lesion. In our patient two previous survey films were supposedly negative, the last one approximately two years before the time of diagnosis. It may be assumed that the lesion must have been much smaller to have been missed even on a small film.

In the case of Ameuille and associates, the lesion had progressed and caused marked bony erosion within a period of eleven years, since on x-ray examination in 1929 the sac was not seen, whereas in 1940 the sac was 8 cm. in diameter and there was extensive bone erosion. In Case 1 of the report of Byron and co-workers, there had been progressive enlargement of the sac over a period of about five years from a 10 cm. diameter to a size where the sac occupied two-thirds of the hemithorax. The common occurrence of erosive bone changes in the reported cases is additional evidence of the progressive nature of this lesion.

Therefore, it is felt that if this lesion is encountered at thoracotomy there should be no hesitancy in treating it surgically by the method appropriate to the individual case. In the cases treated surgically in the present era of thoracic surgery, the early results have been good. The first surgical patient, who was operated upon prior to the present development of intrathoracic surgery, died as a result of empyema and meningitis. The other case which ended in a similar fashion was done in 1940, *but no attempt was made to close the dural defect,* and, in view of this, it is difficult to see how the result could have been otherwise. The remainder of the cases treated by direct surgical treatment of the lesion number five, including our own case. One 61-year-old poor-risk patient died on the table. The other four have shown early favorable results. Obviously the late results, after a period of years to determine the presence of recurrence, will have to be considered before final evaluation of the treatment can be made.

However, with the use of additional aids in closure of the defect when indicated, such as fascia lata, nonreactive polyethylene, absorbable cellulose, grafts, and other modern aids, it seems that a permanent successful result should be obtained.

We have purposely delayed reporting our own case in order to have more than a short postoperative follow-up. At the time of writing, three and one-half years postoperatively, there is no evidence of any recurrence.

*Preoperative Diagnosis.*—When the possibility is suspected, as in a patient with neurofibromatosis, confirmation may be obtained by instillation of radiopaque substance into the spinal canal followed by posturing in an attempt to get the opaque material into the sac and then to demonstrate it roentgenologically. Or the reverse may be done, as demonstrated in one of the cases reported by Byron and associates, in which opaque material was instilled into the sac by direct puncture through the thorax and then made to enter the spinal canal.

Institution of pneumothorax with thoracoscopic visualization, palpation with a rod for consistence, etc., as reported by Ameuille and co-workers, might be done. To this one could add direct thoracoscopic puncture with analysis of the fluid.

Puncture through the thoracic wall is simple and practical, except for the natural reluctance to do this in the presence of a "tumor mass" of unknown nature.

It is felt that the exact preoperative diagnosis of this lesion may not be of such great importance; if there were no contraindication to operation on the basis of a diagnosis of neurofibroma of the mediastinum, it is felt that the same thinking may apply in the case of meningocele, especially if one subscribes to the view that this tends to be a progressive lesion.

## CONCLUSIONS

It is recommended that if encountered at thoracotomy a meningocele should be treated surgically, since the evidence points to the premise that this is a progressive lesion. If the diagnosis is made preoperatively in a patient with symptoms it is felt that surgery is indicated. If asymptomatic, extended roentgenogram observation over a period of years may be used to determine the advisability of surgery.

The surgical treatment should include meticulous closure of the dura. This closure and that of the bony defect should be supported by whatever means possible—parietal pleura, muscle flap, fascia lata, nonreactive polythene, etc.

Four operative cases, including the author's, reported in the American literature, which were done in the era of the present development of chest surgery, have shown early favorable results. In a fifth a mortality occurred during surgery in a poor-risk patient. Two cases reported in the European literature, done at an earlier period, died of infection with meningitis. This brings the total of patients subjected to direct surgical attack of the lesion to seven.

## SUMMARY

1. A case of successful excision of an intrathoracic meningocele is presented. There has been no recurrence during a three and a half year follow-up.
2. Previous surgically treated cases are reviewed and discussed. These number six, the author's case bringing the total to seven.
3. The preoperative diagnosis, surgical indications, and surgical treatment are reviewed.
4. It is felt that this is a progressive lesion and that, in general, surgery is indicated.

## ADDENDUM

Since this paper was written the patient has been seen again and continues to do well with no roentgenographic evidence of recurrence.

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# LACERATION OF THE AORTA

## Case Report of Successful Repair Forty-eight Hours After Injury

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STAB wounds of the great vessels are usually fatal. Bradford and Johnston reviewed the world's literature in 1950, and found that ninety cases of traumatic rupture of the aorta had been reported. Only seven of the ninety patients lived more than eighteen hours from the injury. Only four patients survived. In all four of these patients the laceration of the aorta had occurred inside the pericardium. No patient with laceration of the aorta outside the pericardium survived. However, Bradford and Johnston described a patient of their own who sustained a traumatic rupture of the aorta just proximal to the diaphragm. Five weeks after the original injury, which broke several ribs in the right thorax, the patient suddenly went into shock and was found to have a hemothorax on the right side. When a right thoracotomy was done about eleven weeks after the injury, profuse bleeding was encountered. The bleeding could be controlled only with packing. A second thoracotomy on the right side eighty-one days after injury disclosed the true diagnosis, but not soon enough to save the life of the patient who died on that day. However, the authors felt that there was no way to save the patient. The lesion was inoperable from the right side, and a left thoracotomy would not have been tolerated with such a large right hemothorax.

The first successful closure of a laceration of the aorta was performed by Dshanelidze of Russia in 1922. The patient was a 22-year-old man who received an 8 mm. laceration of the aorta 1 cm. above the heart. No mention was made of the weapon used nor of the time that elapsed before the laceration was closed.

The second case was reported by Blalock in 1932. His patient was an 18-year-old man stabbed with an ice pick about forty-five minutes before admission to the hospital. The size of the laceration was not stated.

The third case was reported by Elkin in 1941. The patient was a 28-year-old woman who was stabbed with an ice pick a few minutes before admission to the hospital. The size of the laceration was not stated.

The fourth case was reported by Elkin in 1944. The patient was a 44-year-old man who was stabbed in the chest with a knife. The patient came to the hospital immediately and was operated upon sixty minutes after admission to the hospital. There was no mention of the size of the laceration.

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### CASE REPORT

A 21-year-old Negro man walked into the Emergency Room of the Gallinger Municipal Hospital at 1:15 A.M., Oct. 31, 1950, with the complaint that he had been stabbed in the chest with a penknife four and one-half hours before. He stated that there had been a moderate amount of bleeding from the wound, that he was slightly short of breath, and that he had pain anteriorly in his left chest on inspiration.

*Family history* was noncontributory except that his grandfather had had a history of convulsive seizures.

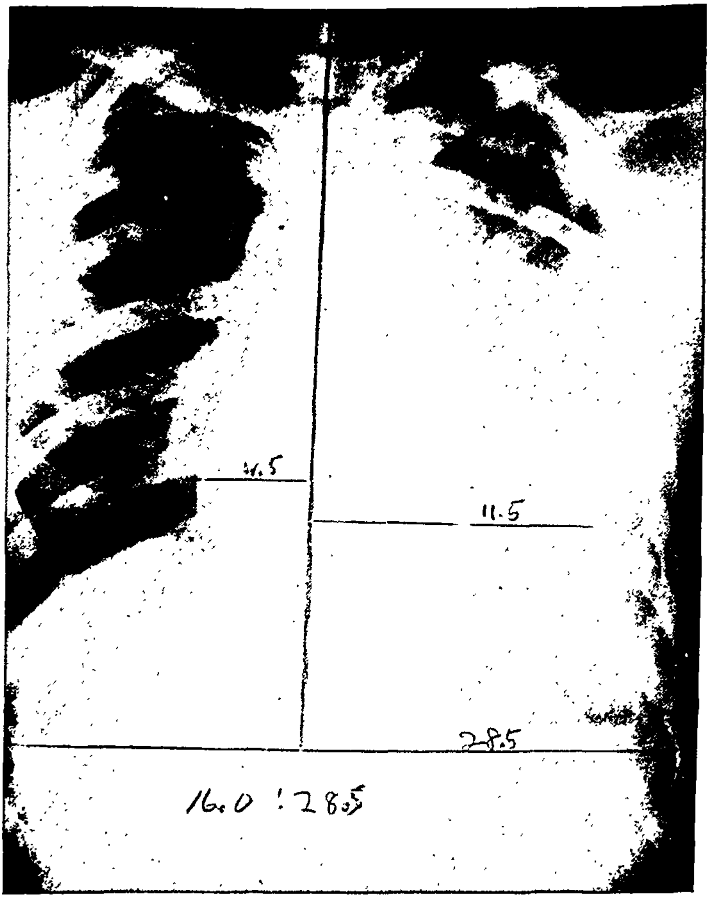

Fig. 1*A*.—Posteroanterior chest roentgenogram taken at the time of admission to the Emergency Room. The knife blade in the mediastinum can be faintly seen.

*Past history* was noncontributory other than that the patient had had a skull fracture at the age of 5 years.

*Physical examination* showed a young Negro man who was moderately dyspneic and uncooperative, but who did not appear to be more than moderately ill. His temperature was 98.6° F. by mouth, pulse rate 100, and respiratory rate 24. The blood pressure in his right arm was 200/120. There was a 2 cm. laceration over the second interspace about 1 cm. to the left of the sternum. The wound bled very slightly. Breath sounds were bilaterally equal and normal. The heart sounds were of good quality, with a short, soft apical systolic murmur. No friction rub was heard. The radial arterial pulse was full and regular.

The remainder of the physical examination was within normal limits.

*Laboratory data:* The hematocrit was 33 and the hemoglobin was 71 per cent. The white blood cell count, the urinalysis, and the serology were normal.

*Hospital course:* A roentgenogram of the chest was made. Wet reading of the film was thought to be within normal limits. The laceration in the skin was sutured, 1,500 units of tetanus antitoxin were administered in the Emergency Room, and the patient was admitted to the hospital on the George Washington Surgical Service.

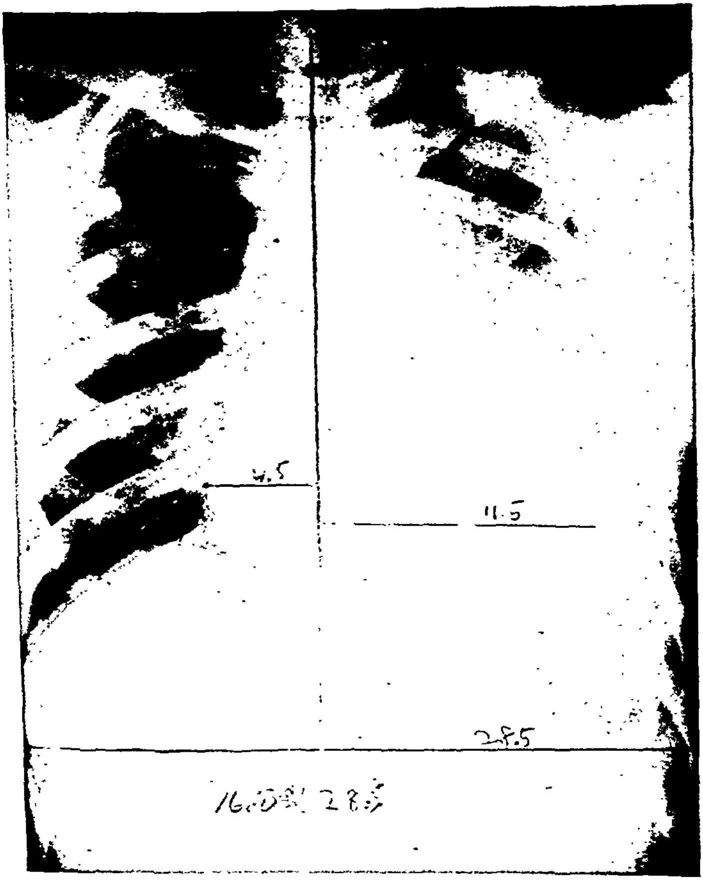

Fig. 1*B*.—This is the same roentgenogram as Fig. 1, but the knife blade has been outlined.

Twelve hours after the injury, the patient had a grand mal seizure. He lost consciousness, had a generalized convulsion, and was incontinent. After this episode, which lasted about two minutes, the patient was drowsy. He was slightly cyanotic after the convulsion, and oxygen administration was started. The neurologic examination was within normal limits but for hypoactive reflexes. The neurologist felt that anoxia had brought latent epilepsy into a grand mal seizure.

The chest roentgenogram taken in the Emergency Room was temporarily misplaced, but it was found several days later. See Figs. 1*A* and *B*. A second chest film was taken thirty-six hours after the injury. This film showed an enlarged cardiac silhouette and a knife blade in the mediastinum. No lateral chest film was taken.

About forty-four hours after the original injury the patient became cyanotic and orthopneic. His blood pressure fell to 70/50. The pulse rate was 140, and it was paradoxical in character. The neck veins were distended. The heart sounds were audible and seemed relatively normal. The liver edge was three fingerbreadths below the costal margin, and the venous pressure was 190 millimeters of saline.

A diagnosis of cardiac tamponade was made, and the patient was prepared for operation.

Approximately forty-five hours after the injury, while preparations for operation were being made, the patient became unconscious and his blood pressure could not be measured. After 30 c.c. of blood were aspirated from the pericardium, he regained consciousness. His blood pressure was then 120/80 and his pulse rate was 120. The patient was still cyanotic and dyspneic.

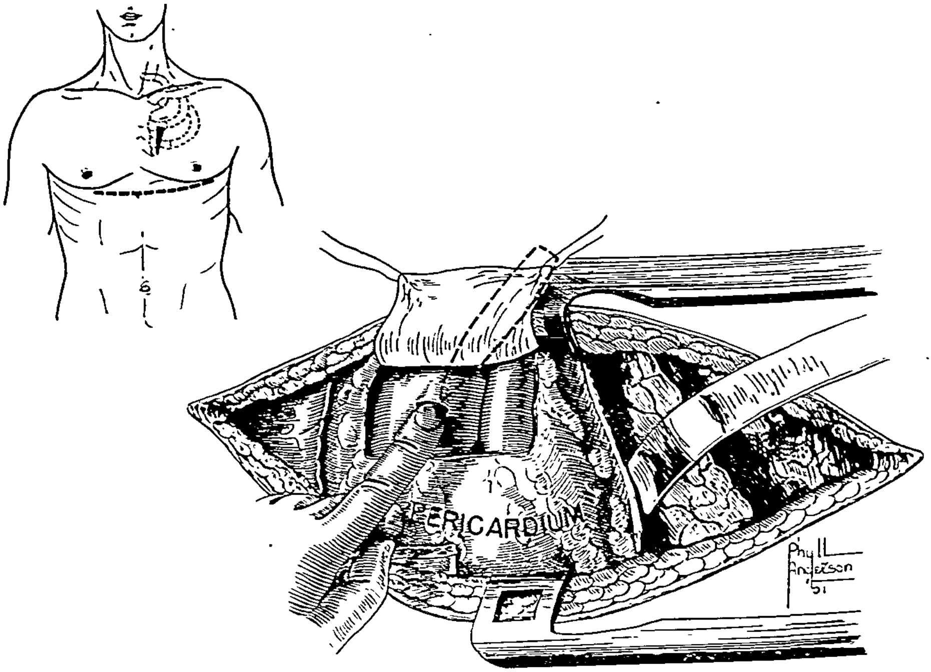

Fig. 2.—A drawing of the operative incision and the appearance of the laceration in the aorta.

*Operation:* The operation was begun nearly forty-eight hours after the injury with pentothal and endotracheal ether anesthesia, and the left chest was opened anteriorly by resecting the left fifth rib from the sternum to the posterior axillary line. About 500 c.c. of blood were removed from the left pleural cavity, but the lung was entirely normal. The anterior mediastinum was hemorrhagic and edematous. Inspection of the heart showed marked muffling of the heartbeat. The incision was carried across the sternum, and the right fifth interspace was incised intercostally for approximately four inches. The pericardium was opened transversely. A thin layer of blood was found to tamponade the heart. Just after the pericardium was opened, bright red blood gushed out from the upper pericardial sac. Immediate digital palpation along the anterior surface of the ascending aorta revealed a laceration about 14 mm. long just inside the superior pericardial attachment. (See Fig. 2.) Hemostasis was maintained with the palpating finger. Each time the patient breathed the point of the knife blade struck the back of the finger compressing the laceration. The aorta was compressed dorsally, the tip of the knife blade was grasped with an instrument, and the blade was withdrawn. It was later found to be 6 cm. in length.

To gain exposure the pericardium was incised in a cephalad direction at each end of the transverse incision, and a pericardial flap was turned upward. Five interrupted 0000 atraumatic silk sutures were used to close the laceration. The left pleural cavity was drained with an intercostal catheter, and the chest was closed in layers with interrupted silk sutures.

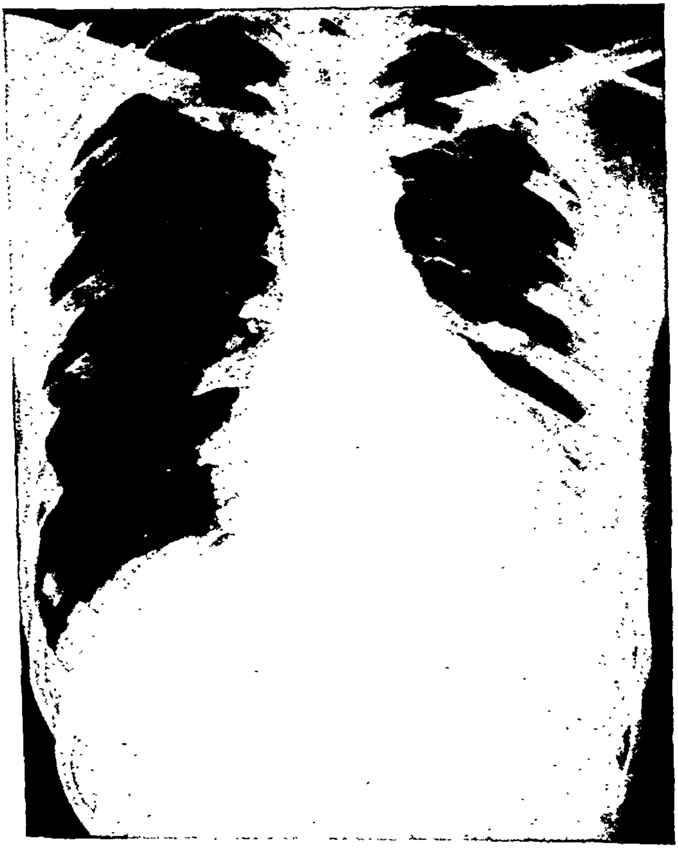

Fig. 3.—A chest roentgenogram taken fourteen days after the operation.

During the operation the blood pressure was at all times above 120 mm. Hg systolic. The blood pressure at the conclusion of the operation was 155/95. The patient received 1,800 c.c. of whole blood and 1,000 c.c. of dextrose solution intravenously during the operation.

*Postoperative course:* The patient was given an additional 500 c.c. of whole blood on the first postoperative day. Penicillin was administered for seven days. On the eighth postoperative day the blood pressure was 118/66. He was discharged to his home on the eighteenth postoperative day (Fig. 3). Three months after the operation the patient was alive and well.

## SUMMARY

A case has been presented in which a knife blade 6 cm. long was removed from the anterior mediastinum forty-eight hours after injury. A laceration of the ascending aorta inside the pericardium was successfully closed. This is the fifth successful repair of a lacerated aorta.

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# ARTERIOVENOUS FISTULA OF THE AORTIC ARCH

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IN NOVEMBER, 1950, Proctor[1] reported the first case of arteriovenous fistula of the aortic arch. This fistula was due to trauma and was successfully repaired.

We have recently had the opportunity to treat a patient with a traumatic arteriovenous aneurysm between the aortic arch and the left innominate vein. In this case the aorta and the vein were not in contact, and there was a definite false sac interposed between the two structures.

As the patient was treated on the same service for the original stab wound which produced the fistula, we had the opportunity to see the heart enlarge due to the effect of the fistula, and then return to normal size after closure. We feel that too long an interval occurred between the onset of the fistula and its closure, but that we were fortunate in getting her to surgery before evidence of cardiac failure appeared.

## CASE REPORT

A 25-year-old Negro woman was admitted to the Shreveport Charity Hospital on Nov. 16, 1949, having been stabbed several minutes before admission. She was in deep shock, with little evidence of external bleeding. There was a 1 cm. stab wound over the left sternoclavicular joint, and several small stab and incised wounds of the arms.

Following a transfusion of 1,000 c.c. of blood, her blood pressure rose to 118/70, although her pulse remained around 120. The next day a continuous murmur with systolic accentuation was heard over the entire chest, being more marked over the aortic area. During the next few days the murmur became louder and a palpable thrill appeared. On Nov. 18, 1949, chest roentgenogram showed a homogeneous density involving the superior mediastinum. The roentgenogram was repeated on Dec. 2, 1949, and showed some resolution of the mediastinal shadow, but the mediastinum was still widened, and there was a definite border extending to the left of the manubrium. An electrocardiogram on Nov. 22, 1949, showed sinus tachycardia. Sound tracings revealed a continuous murmur, with systolic accentuation, appearing to be of greatest intensity on the medial side of the wound. Next in intensity was the area on the lateral side of the wound, with the inferior area being next in intensity, and the superior area being least in intensity. Blood pressure was equal in both arms on several occasions. One venous pressure determination showed a pressure of 110 mm. of water in the left ante-cubital vein, and 140 mm. in the right ante-cubital vein. At this time, however, the right arm was somewhat swollen and edematous due to reaction around its laceration. She ran a febrile course spiking to 100° F. for one week, and then became afebrile. At the time of her discharge on Dec. 5, 1949, she was asymptomatic except for consciousness of the murmur and thrill.

On her return admission on Jan. 1, 1950, the electrocardiogram was normal, the rate being seventy-five per minute. Roentgenogram of the chest showed almost complete resolution of the superior mediastinal shadow. There was some increase in the size of the heart. The loud, machinelike murmur with systolic accentuation was still present, as was the thrill. She was discharged to the clinic on Jan. 9, 1950, to be admitted for surgical correction of the fistula within three or four months.

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She was readmitted, having been lost to follow-up in the meantime, on Nov. 24, 1950, with no symptoms other than an awareness of the thrill, of which she was constantly conscious. Blood pressure on admission was 106/50 in the right arm, and 112/60 in the left. The murmur was loud and heard all over the chest and back, but was loudest over the manubrial area. The palpable thrill was also more noticeable over this area. Roentgenogram of the chest showed some widening of the superior mediastinum and a definite cardiac enlargement, confirmed by fluoroscopy, since the last admission. Electrocardiogram was within normal limits.

The operation was carried out Dec. 3, 1950. The incision described by Shumacker[2] was used, and gave excellent exposure. The incision started at the middle of the left clavicle, curved down over the left sternoclavicular joint, and extended down to the midsternum. The veins of the anterior chest wall and neck were extremely dilated. The inner one-half of the left clavicle was resected subperiosteally and the attachments of the sternomastoid, sternohyoid, and the sternothyroid muscles were sectioned. This exposed a tremendously dilated innomonate vein through whose wall blood could be seen whirling in eddies. The retrosternal space was entered with difficulty because of previous reaction and the presence of a pulsating mass beneath it.

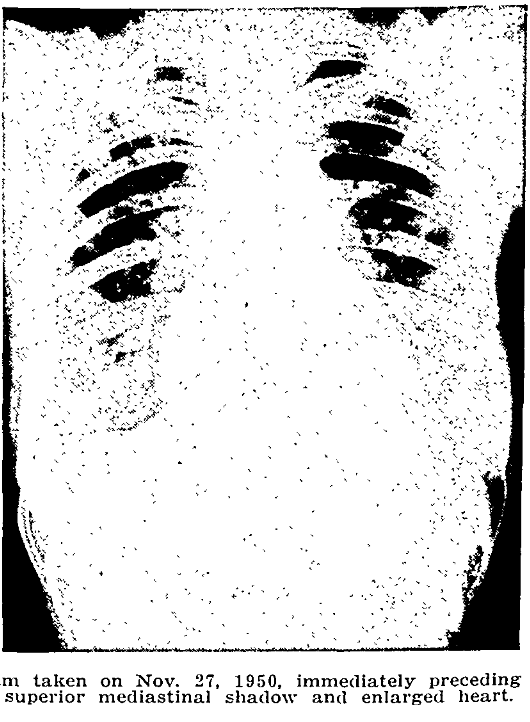

Fig. 1.—Roentgenogram taken on Nov. 27, 1950, immediately preceding the operation. Note superior mediastinal shadow and enlarged heart.

It was necessary to split the sternum considerably to the right of the midline to avoid this mass. The sternum was split down to the third interspace with the Lebsche Sternum Knife, and then cut through on each side. The left part was elevated with bone hooks and separated from the underlying mass by sharp dissection. A small rib spreader then gave excellent exposure.

A pulsating, thick-walled mass about 8 cm. in diameter occupied the entire retrosternal space. The dilated left innominate vein crossed superior to this mass and was firmly attached at this point. Pressure with the finger here stopped the visible flow of arterial blood into the vein. The vein was dissected free and rubber dam drains placed around it, both proximal and distal to its communication with the sac.

The sac was then dissected free with considerable difficulty, but there was no significant bleeding. It was necessary to mobilize, but not remove, the thymus, and dissect off both anterior pleural reflections. Neither pleural cavity was entered. The sac was completely mobilized and was found to communicate with the aorta on its anterior-superior surface, about 2 cm. distal to the origin of the innominate artery.

Fortunately the sac narrowed at this communication and connected to the aotra by a neck about 3 cm. in length and 2 cm. in diameter. A rubber drain was passed around the aortic communication and it was occluded. This stopped the thrill and had no effect on the pulse of either arm or either cartoid artery. The rubber drains around the vein were then occluded and the aneurysm opened and emptied. There was no bleeding.

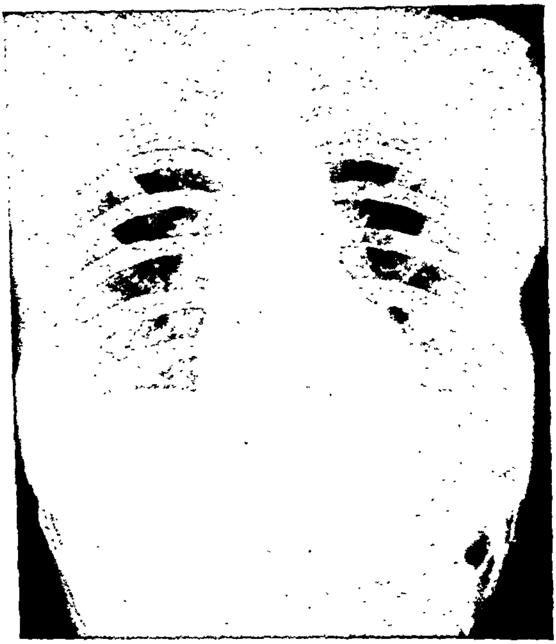

Fig. 2.—Roentgenogram taken on March 9, 1951. Note absence of superior mediastinal shadow, decrease in heart size, and regenerating left clavicle.

The opening into the innominate vein was about 2 cm. long and slitlike. While feasible, suture of this opening did not appear necessary, so the innominate vein was ligated and suture ligated on each side of the communication and severed, leaving a small segment attached to the sac. The sac was then elevated and the aortic communication ligated with No. 2 braided silk flush with the aortic wall. A suture ligature of the same material was placed distal to the ligature and the sac completely excised. The aortic communication was further closed with interrupted No. 00 cotton sutures. The sternum was brought together with periosteal sutures, and the wound closed, placing bone chips in the bed of the resected clavicle.

Five hundred cubic centimeters of blood were given during the operation.

The postoperative course was relatively uneventful, except for a febrile reaction to 102° F. on the first postoperative day. This gradually subsided, the wound healed well, and she was discharged Dec. 17, 1950, on her twelfth postoperative day.

When last seen on March 9, 1951, she was *asymptomatic*, no murmur was present, arm and shoulder functions were *excellent*, and she had resumed work. Roentgenogram of the chest at this time showed no discernible widening of the mediastinum, beginning regeneration of the clavicle, and a heart that had returned to normal size.

## CONCLUSION

1. The second case of arteriovenous fistula of the aortic arch is presented.
2. This has apparently been cured by the removal of a false aneurysmal sac between the arch of the aorta and the left innominate vein.

## ADDENDUM

This patient was seen June 12, 1951. At this time she was asymptomatic, and no murmur or thrill was present. Roentgenogram of the chest was within normal limits. Clinically there was firm regeneration of the clavicle and arm function was excellent.

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1. Proctor, William H., Jr.: Arteriovenous Fistula of the Aortic Arch, J. A. M. A. 144: 818-819, 1950.
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# AN EXPERIMENTAL EVALUATION OF CATGUT AS A VASCULAR SUTURE MATERIAL AND A REPORT ON ITS CLINICAL USE

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CERTAIN developments occurring during little more than the past decade have caused what might be considered the renaissance of vascular surgery. The great success enjoyed in the extension of surgical treatment of congenital anomalies of the heart and great vessels, and in the care of vascular injuries of World War II, have stimulated laboratory studies to the extent that nearly all centers of medical research are now pursuing one or more major projects related to vascular surgery.

As might be expected, certain of the time-honored and traditional rules governing vascular surgical technique, as evolved at the beginning of the century, have been greatly modified. The advent of anticoagulant and antimicrobial drugs, the development of large blood banks, and the use of intra-arterial transfusion have been major factors in this metamorphosis.

Opposition to the use of catgut as a vascular suture developed in the early years when infection, thrombosis, and hemorrhage were common complications of major vascular surgery. This seems to be almost a paradox, since catgut was the standard suture material of the time, and only a few pioneer surgeons such as Kocher[1, 2] and Halsted[3, 4] were showing real interest in silk thread. While subsequent years saw a slow, steady shift in favor of fine, nonabsorbable suture material, the use of fine silk in vascular surgery had long been firmly established.

Although the controversy of silk versus catgut continued to rage in general surgery (Halsted,[3-4] Haegler,[5] Heidenhain,[6] Howes,[7-11] Whipple,[12] Elkin,[13] Bower and Pearce,[14] and Gage and Lyons[15]) there appeared to be no disagreement among vascular surgeons as to the superiority of silk. However, as increasing numbers of children were operated upon for coarctation of the aorta, tetralogy of Fallot, and portal hypertension where growth at the site of anastomosis commensurate with that of the individual would be desirable, experimental studies have appeared, reporting the relative value of various techniques of vascular suture and kinds of suture material, especially in the growing animal.[16-22a] It was shown by certain investigators that fine chromic catgut was a more satisfactory vascular suture material than silk when used as a continuous suture in a growing animal.[18, 19, 20] These observations were derived, however, from the study of single anastomoses in the repair of the aorta of dogs and pigs. We considered it possible to obtain a better comparison of vascular healing, if

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## CONCLUSION

1. The second case of arteriovenous fistula of the aortic arch is presented.

2. This has apparently been cured by the removal of a false aneurysmal sac between the arch of the aorta and the left innominate vein.

## ADDENDUM

This patient was seen June 12, 1951. At this time she was asymptomatic, and no murmur or thrill was present. Roentgenogram of the chest was within normal limits. Clinically there was firm regeneration of the clavicle and arm function was excellent.

## REFERENCES

1. Proctor, William H., Jr.: Arteriovenous Fistula of the Aortic Arch, J. A. M. A. 144: 818-819, 1950.
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preparations were made of sagittal sections of the entire graft and adjacent host vessel. Hematoxylin and eosin, Voerhoff's elastic stain and trichrome connective tissue stains were used in all instances.

## RESULTS

*Technical Evaluation of Suture Material.*—It was a unanimous opinion among five vascular surgeons at Presbyterian Hospital testing both sizes of catgut and silk, that the latter seemed easier to handle, although others have felt the reverse to be true.[14] One of us who had previously used catgut extensively in general surgery concurred in this opinion, although the other operators had always used silk as a standard suture material. The gut was stiffer and appeared to dry quickly, especially when used as a traction suture. Furthermore, the tensile strength on occasion appeared to vary in a single strand, with unexpected breakage or fraying occurring while performing an anastomosis.

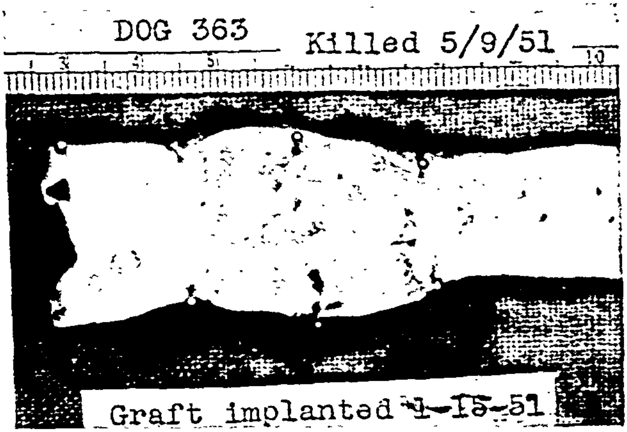

Fig. 1.—Graft obtained from thoracic aorta and stored in a nutrient electrolyte solution seven days before use in thoracic aorta of another dog. After implantation of one hundred fourteen days, the proximal chronic catgut anastomosis (left) is barely visible, whereas a few silk sutures mark the distal suture line. The white spots are highlights.

Since the usual vascular anastomosis depends upon one suture line, this apparent lack of uniformity gave some concern, and has been noted by others.[22, 23] While we have encountered similar variability in some commercial brands of braided arterial silk, the lots of silk used in this study were very satisfactory in all respects.

*Function and Gross Appearance of the Implanted Grafts and Anastomoses.*—The eight grafts used in the abdominal aorta had been quick frozen at –72° C. and stored at low temperatures. Four had been kept for from twenty-three to one hundred thirty-five days at –27° C. and four for from twenty-three to ninety days at –70° C. (Table I.) One partial occlusion by a mural thrombus occurred in the former group. In this one, there was a moderate sized pedunculated thrombus originating from the proximal catgut suture line, but it was well endothelized and did not interfere with function significantly. Among chromic catgut and silk were used in the same animal for the vascular anastomoses. By implantation of carotid and aortic grafts, the suture material could be studied when used with viable and nonviable grafts and in vessels of varying size. The anastomosis of a nonviable homograft to an adjacent normal vessel under tension should impose a significant stress on a suture material.

## METHODS

A total of twenty dogs were used in this study. One group of twelve adult mongrel dogs were used to study the suture lines of aortic grafts. Of these, eight had implantation of the graft into the abdominal aorta distal to the renal arteries, and four into the descending arch of the thoracic aorta. In all of the operations on the abdominal aorta, a comparable segment of normal aorta was excised before implanting the graft. The grafts measured 1.5 to 3.2 cm. in length and had been stored for periods up to six months (Table I). Only during the implantation of the thoracic grafts was a paraffin-lined glass tube used for temporary restitution of aortic blood flow. The tube was removed just before completion of the distal anterior suture line. By this method, the time during which the aortic blood was halted was two periods of about three minutes each. In none of these dogs nor in any of the others was there any muscular weakness or paraplegia.

A group of eight adult mongrel dogs were used to study anastomosis of carotid grafts. Both arteries were exposed by a vertical incision in the neck. In all instances, a segment 1.5 to 2.9 cm. in length was excised from the mobilized right common carotid artery and implanted into the left common carotid artery, after excision of a segment of comparable length. A carotid homograft measuring about 2.0 cm. and which had been preserved for up to fifteen weeks was then implanted into the defect in the right common carotid.

The proximal and distal anastomoses were accomplished in the carotid vessels by the Carrel technique. In the aorta, an everting continuous horizontal mattress suture was employed. Two single horizontal mattress traction sutures were placed equidistant along the circumference. The posterior row was completed by rotation of the vessel and graft. After running the free suture from one traction suture to the other and tying them together, the vessel was rotated back into normal position and the anterior row completed in a similar manner, using the free end of the second traction suture. In all the grafts nonboilable medium chromic catgut swaged on a one-half circle atraloc taper point needle[*] was used for the proximal anastomosis. For the inferior or distal anastomosis, braided black silk swaged on a three-eighths circle taper point needle[†] was employed. For implanting the carotid grafts, we used 000000 suture material. whereas we preferred 00000 for the aortic anastomoses.

Anticoagulant drugs were not given to any of these animals. The function of the graft was checked frequently by evaluation of the peripheral arterial pulses, and at rare intervals by angiography.

When the animals died or were killed, the graft was excised with adjacent host vessel and was opened by an incision in its long axis. The suture line was observed for constriction or thrombosis and for the amount of suture material presenting on the inside of the lumen. Of secondary interest were any degenerative changes or localized thrombi involving the actual graft. Microscopic beneath the endothelium became greater as the eversion of the cut edges of the vessels produced by the mattress sutures became less (Fig. 3). After three or four months, the eversion is often barely discernible grossly and photomicrographs may show essentially end-to-end healing of the edges of the vessels.[18] As this flattening occurs, the suture material slowly assumes a superficial vertical alignment. As this process develops in the eversion type of anastomosis, lengths of suture material of one to three millimeters may be seen at the site of each stitch. This appearance should not be attributed to faulty suture technique, as has at times been claimed. In this respect, the catgut anastomosis always appeared better than the silk one since the absorbable material often was not grossly visible after several weeks (Fig. 3). Function was equally satisfactory, however.

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[*] Supplied by Ethicon Suture Laboratories, Inc., Type C 5-0 Code F131; Type C 6-0 Code F130.

[†] Supplied by J. A. Deknatel & Son, Inc., 5-0 Lot No. 155; 6-0 Lot No. 156.

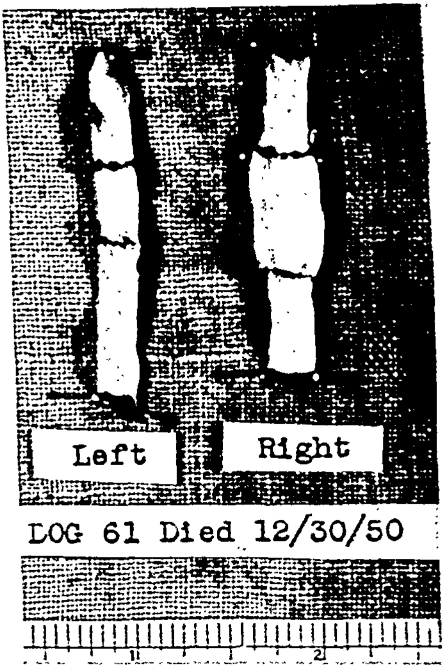


Fig. 2.—The autograft in the left carotid artery reveals no gross differences from the recipient vessel. The homograft implanted into the right carotid artery after three weeks of storage at −70° C. is slightly thinned and dilated. The suture lines appear very satisfactory, with chromic catgut still intact in the lower anastomoses, twenty-five days after implantation.

Minor constriction at the everted anastomosis was observed in a few instances at the close of operation. Subsequent study of the anastomoses to determine the degree of constriction revealed no constriction in any of the catgut series greater than that observed at operation. After three or four weeks, even this minor constriction had become less evident. This effect coincided with absorption and fragmentation of the catgut suture material. In the silk group of anastomoses, the degree of constriction at three to six weeks was essentially the same as at time of surgery, or slightly greater. Later, however, flat-the others, examined from seven to ninety-three days after implantation, three small endothelized mural thrombi were found at necropsy. Function had been excellent in all.

Of the four segments of aorta implanted into the thoracic aorta, one had been quick frozen at –72° C. and stored for one hundred and seventy-eight days at –70° C., whereas the other three had been refrigerated at 4° C. in Simm's X6 solution containing 10 per cent homologous serum for seven, sixteen, and two hundred and four days. In none of these did thrombus formation occur (Fig. 1). With one exception the anastomoses were examined after periods of implantation of over one hundred days.

The carotid autografts implanted in eight dogs retained the gross appearance and physical characteristics of the normal adjacent vessel. In none of the specimens was there any visible degeneration or thrombus after implantation of from sixteen to one hundred and sixty-eight days. It was interesting to note that the minimal fibrous reaction binding the graft to the adjacent tissues of the neck was often of about the same degree observed on the side with the carotid homograft.

Of the eight carotid homografts, four were frozen grafts stored at –70° C. for eighteen to fifty-five days, and four were grafts kept in the nutrient solution for from thirteen to fifty days. In one from each group there was complete occlusion by thrombus formation. In another of the frozen grafts, function was diminished by a thrombus. All of these carotid grafts with thrombosis had been stored six weeks or longer (Table I). The inside diameter was generally about three millimeters. These grafts were examined after from sixteen to one hundred and sixty-eight days of implantation. The homografts of both types were thinner, less elastic, and slightly dilated when compared to the autograft in the opposite carotid artery (Fig. 2) until two to three weeks, after which the wall was thickened by fibroplasia and dilatation appeared diminished

In no instance was there a dehiscence of the suture line. Two animals had old hematomas adjacent to a carotid graft but no evidence of any source of leakage from either suture line was apparent at the time of operation or autopsy.

The suture lines of the twenty homografts and eight autographs were examined from one day to one hundred and sixty-eight days after anastomosis. The early gross appearance was very comparable, with little or no suture material visible in the lumen. *An equivalent minor degree of inflammatory* response was noted at both of the suture lines, with endothelization occurring within a few days in all instances. As noted before, sizable thrombi involved the proximal catgut suture line and the homograft in one aorta and three carotid arteries. Three additional abdominal aortic grafts had sessile mural thrombi limited to the mid-portions, several millimeters from either suture line. In none of the autografts was there any thrombus.

At about two weeks, or even less, *minimal vertical lengths of catgut* or silk suture material were visible under the new endothelium at the line of anastomosis. Little more suture material became visible subsequently in the carotid specimens in which a continuous Carrel technique had been employed (Fig. 2). However, in the aortas, the visible lengths of silk and catgut sutures tening of the eversion in many permitted some diminution of the constriction. In none of the grafts was the functional capacity altered by the minimal constriction present at the anastomoses.

*Microscopic Appearance of the Grafts and Anastomoses.*—This section deals primarily with the histologic changes observed at the suture lines rather than with those occurring in the actual grafts. The latter findings have been described in detail in other publications of ours.[24, 25, 26]

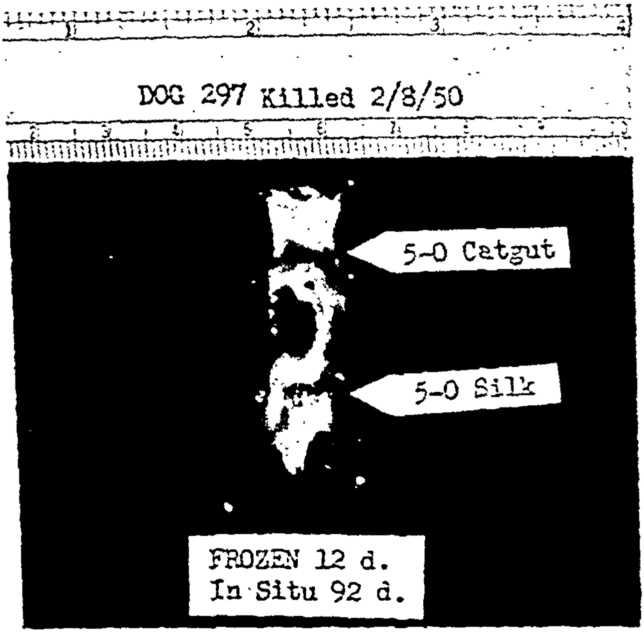


Fig. 3.—Graft obtained from the abdominal aorta and stored twelve days at -27° C., before implantation into the abdominal aorta of another dog for ninety-two days. There is an erosion of the intima in the midportion of the graft, but no thrombosis. The chromic catgut is not recognizable grossly, but the silk sutures are visible beneath the endothelium covering the distal anastomosis.

It will suffice to reiterate that all of the homografts, whether preserved by freezing or in a nutrient solution, and whether of aorta or carotid artery, undergo a slow degeneration during storage. The endothelium and many of the smooth muscle cells have disappeared by the time the graft is used, and usually within several weeks after implantation, even the residual cellular elements of the graft have disappeared. This process is somewhat slower in grafts stored in nutrient solution. The grafts become lined as well as invested externally by thick fibroblastic layers from the host within days, and after several months the persisting elastic and collagen framework of the graft itself are infiltrated with the new fibroblasts. Observations carried out for as long as two years following implantation reveal that the original elastic framework remains grossly intact, although some fragmentation of the fibers can usually be detected, whereas the remainder of the homograft has become replaced by fibrous tissue from the recipient animal.[26]

TABLE I

| TYPE OF GRAFT | DOG NO. | AGE OF GRAFT (DAYS) | LENGTH OF GRAFT | METHOD OF STORAGE (CM.) | DURATION IMPLANTED (DAYS) | FUNCTION | STATUS OF GRAFT EXCISED |  |  |  | CAUSE OF DEATH | WEIGHT OF DOG (KG.) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  |  |  |  |  | THROMBOSIS | EROSION | DILATED | THINNED |  |  |
| *Aortic:*<br>Abdominal<br>Frozen | 232 | 23 | 2.5 | -27° C. | 7 | Excellent | 0 | + | + | + | Killed | 19.9 |
|  | 212 | 64 | 2.6 | -27° C. | 12 | Excellent | + | 0 | + | ± | Distemper | 9.9 |
|  | 433 | 135 | 3.2 | -27° C. | 12 | Good | +* | 0 | + | + | Killed | 12.1 |
|  | 258 | 45 | 2.2 | -27° C. | 61 | Excellent | 0 | + | 0 | 0 | Killed | 12.2 |
|  | 435 | 35 | 2.7 | -70° C. | 7 | Excellent | + | 0 | + | + | Killed | 11.1 |
|  | 476 | 42 | 2.6 | -70° C. | 14 | Excellent | + | 0 | ± | ± | Killed | 13.0 |
|  | 227 | 12 | 1.5 | -70° C. | 92 | Excellent | 0 | + | 0 | 0 | Killed | 12.3 |
|  | 215 | 48 | 2.5 | -70° C. | 93 | Excellent | 0 | 0 | 0 | 0 | Killed | 14.6 |
| Thoracic<br>Frozen | 360 | 178 | 2.4 | -70° C. | 138 | Excellent | 0 | 0 | 0 | 0 | Killed | 15.2 |
| Nutrient | 380 | 16 | 3.0† | Simms X6 | 1 | Excellent | 0 | 0 | 0 | 0 | Shock | 12.8 |
|  | 87 | 204 | 3.2 | Simms X6 | 100 | Excellent | 0 | 0 | 0 | 0 | Killed | 15.2 |
|  | 363 | 7 | 3.2 | Simms X6 | 114 | Excellent | 0 | 0 | 0 | 0 | Killed | 10.8 |
| *Carotid artery:*<br>Homologous<br>Frozen | 27 | 18 | 2.4 | -70° C. | 16 | Excellent | 0 | 0 | + | + | Killed | 9.8 |
|  | 61 | 22 | 1.7 | -70° C. | 25 | Excellent | 0 | 0 | + | + | Distemper | 9.8 |
|  | 51 | 46 | 2.9 | -70° C. | 109 | Good | +* | 0 | 0 | 0 | Distemper | 15.2 |
|  | 53 | 55 | 2.1 | -70° C. | 113 | Occluded | +* | 0 | 0 | 0 | Killed | 12.2 |
| Nutrient | 26 | 13 | 2.3 | Simms X6 | 21 | Excellent | 0 | 0 | + | + | Distemper | 16.7 |
|  | 63 | 15 | 1.8 | Simms X6 | 31 | Excellent | 0 | 0 | ± | ± | Distemper | 9.8 |
|  | 50 | 43 | 2.9 | Simms X6 | 104 | Occluded | +* | 0 | 0 | 0 | Killed | 19.2 |
|  | 52 | 50 | 2.1 | Simms X6 | 168 | Excellent | 0 | 0 | 0 | 0 | Killed | 12.8 |
| Autologous grafts | 27 | 0 | 2.3 | None | 16 | Excellent | 0 | 0 | 0 | 0 |  |  |
|  | 26 | 0 | 1.5 | None | 21 | Excellent | 0 | 0 | 0 | 0 |  |  |
|  | 61 | 0 | 2.0 | None | 25 | Excellent | 0 | 0 | 0 | 0 |  |  |
|  | 63 | 0 | 1.7 | None | 31 | Excellent | 0 | 0 | 0 | 0 |  |  |
|  | 50 | 0 | 1.5 | None | 104 | Excellent | 0 | 0 | 0 | 0 |  |  |
|  | 51 | 0 | 2.9 | None | 109 | Excellent | 0 | 0 | 0 | 0 |  |  |
|  | 53 | 0 | 1.9 | None | 113 | Excellent | 0 | 0 | 0 | 0 |  |  |
|  | 52 | 0 | 1.9 | None | 168 | Excellent | 0 | 0 | 0 | 0 |  |  |

*Moderate to large thrombus reducing or blocking blood flow through the graft.

†Subclavian artery implanted into graft.

tening of the eversion in many permitted some diminution of the constriction. In none of the grafts was the functional capacity altered by the minimal constriction present at the anastomoses.

*Microscopic Appearance of the Grafts and Anastomoses.*—This section deals primarily with the histologic changes observed at the suture lines rather than with those occurring in the actual grafts. The latter findings have been described in detail in other publications of ours.[24, 25, 26]

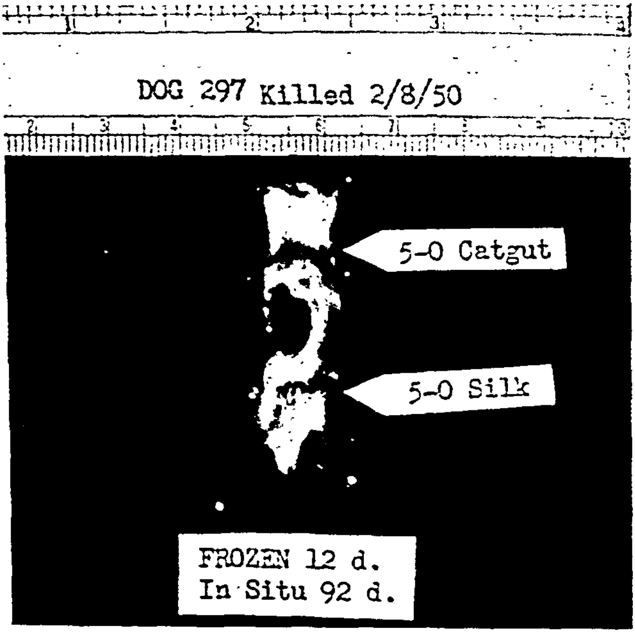
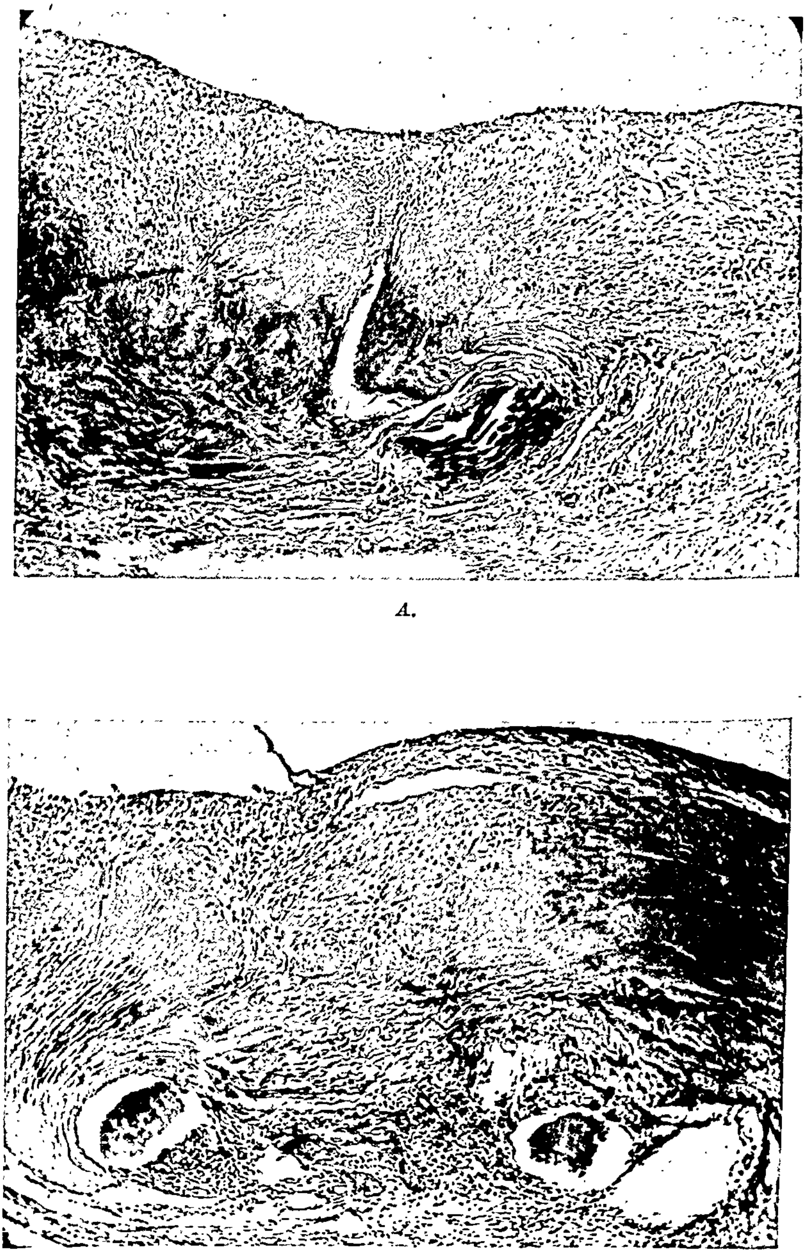


Fig. 3.—Graft obtained from the abdominal aorta and stored twelve days at −27° C., before implantation into the abdominal aorta of another dog for ninety-two days. There is an erosion of the intima in the midportion of the graft, but no thrombosis. The chromic catgut is not recognizable grossly, but the silk sutures are visible beneath the endothelium covering the distal anastomosis.

It will suffice to reiterate that all of the homografts, whether preserved by freezing or in a nutrient solution, and whether of aorta or carotid artery, undergo a slow degeneration during storage. The endothelium and many of the smooth muscle cells have disappeared by the time the graft is used, and usually within several weeks after implantation, even the residual cellular elements of the graft have disappeared. This process is somewhat slower in grafts stored in nutrient solution. The grafts become lined as well as invested externally by thick fibroblastic layers from the host within days, and after several months the persisting elastic and collagen framework of the graft itself are infiltrated with the new fibroblasts. Observations carried out for as long as two years following implantation reveal that the original elastic framework remains grossly intact, although some fragmentation of the fibers can usually be detected, whereas the remainder of the homograft has become replaced by fibrous tissue from the recipient animal.[26]



*A.*

*B.*

Fig. 4.—Photomicrographs of the suture lines of a carotid homograft stored twenty-two days at −70° C. and functioning for twenty-five days in another dog. (Hematoxylin and eosin, ×100.) *A*, The silk suture material is unaltered. A mild cellular reaction is still evident adjacent to the silk. Firm fibrous union and smooth endothelization has occurred. *B*, The chromic catgut is being removed by macrophages. A moderate cellular reaction surrounds the suture material. Firm fibrous union and smooth endothelization has occurred. The acellular frozen graft is visible to the right beneath the fibrocellular intimal zone derived from the host vessel.

The autologous graft, as represented by the segment of carotid transferred to the contralateral artery, revealed very few histological changes following transplantation.

Study of the anastomoses by histological preparations suggested that some of the early inflammatory response observed was evoked by the homograft, rather than by the suture material. Polymorphonuclear leukocytes appeared at the anastomosis and external surface of the graft within hours. At one day, there was noted a fresh thrombus in the wedge-shaped cleft on the luminal side of the anastomosis, produced by eversion of the edges of the vessels. The fibrin matrix of this plug served to allow bridging of the anastomosis by endothelium and fibroblasts from the host aorta within a week. Since the homografts were usually devoid of endothelium, a thickening fibrocellular layer developed over the anastomosis by two or three weeks, as the new intimal lining advanced toward the midzone of the graft from each end.

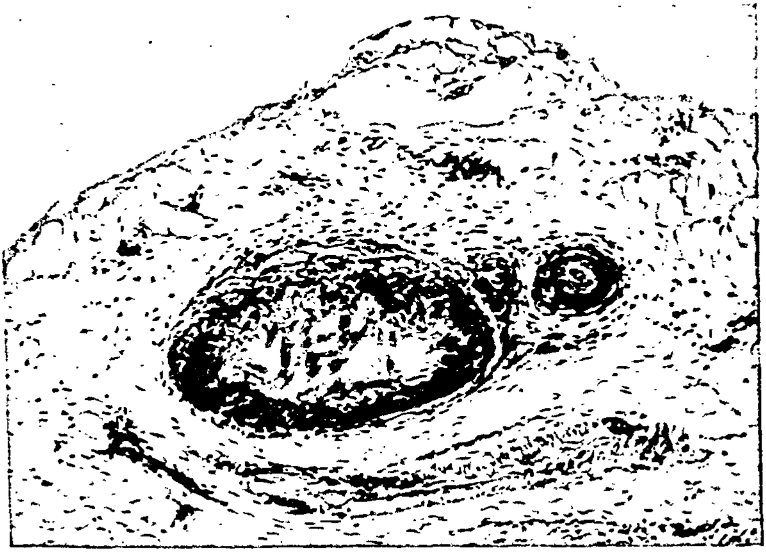


Fig. 5.—Granulomatous reaction surrounding silk suture in an autologous carotid graft, twenty-five days following anastomosis.

At the anastomosis, round cells and macrophages appeared within a few days, this time evoked apparently by the suture material which acted as a foreign body. In some specimens it appeared that the cellular response about the suture material was greater with catgut during the first week, but the differences often were not striking. Some individual variation in animals was noted insofar as cellular response to suture material. In some there was minimal reaction about both catgut and silk, whereas in others there was a moderate to marked inflammatory reaction about both (Fig. 4, *A* and *B*). This fact has been overlooked by many investigators studying suture materials by using each type alone in comparable groups of animals. After ten days to two weeks, the inflammatory cellular reaction about the suture material usually diminished markedly. Macrophage activity was variable in the different animals, generally being most predominant about the catgut.

By one month the tissues about the silk were usually free of cellular reaction, but in some specimens, granulomas developed about the suture strands (Fig. 5). By this time, the tissues about the catgut were also free of cellular reaction except for scattered macrophages, and the suture, staining as hyaline basophilic material, appeared to be diminishing in size (Fig. 6). Firm healing had occurred by fibrous union and a multilayered intima covered the anastomosis. The graft and normal aorta were compressed where eversion occurred, and at times appeared acellular at the point of approximation, where compression was greatest (Fig. 7, *A* and *B*).

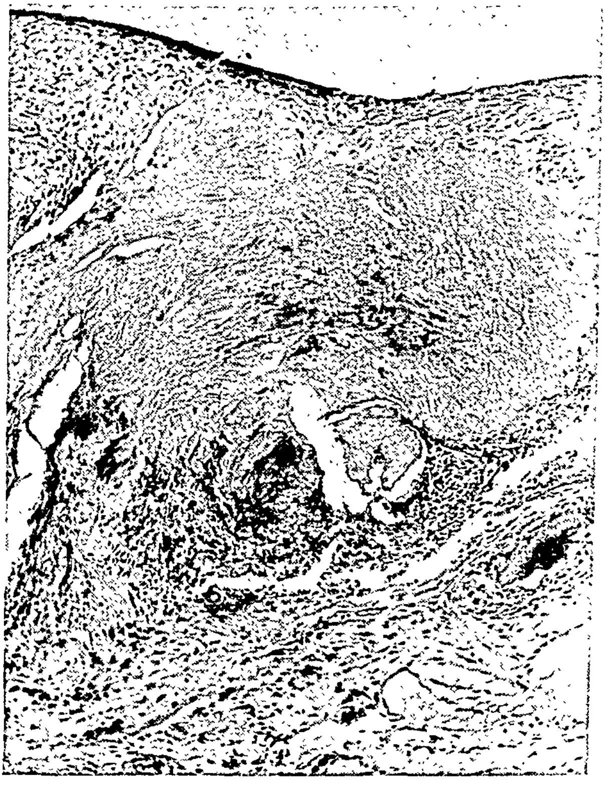


Fig. 6.—Anastomosis of carotid graft preserved fifteen days at 4° C. in a nutrient solution. At thirty-one days chromic catgut still remains beneath a fibrous union. The minimal cellular reaction consists mainly of round cells and macrophages.

During subsequent months, the status of the silk remained essentially unchanged. The catgut was recognizable without difficulty after eight to ten weeks in most specimens; moreover, in a few, the suture strand was only slightly altered at three months. After fragmentation and removal by macrophages had occurred, the site of the suture was identified only by a punctate fibrous scar.

*Clinical Application.*—Because of the satisfactory experimental results with fine medium chromic catgut as a vascular suture material, it was considered as safe for anastomoses of vessels in infants or children in whom growth is the major factor.

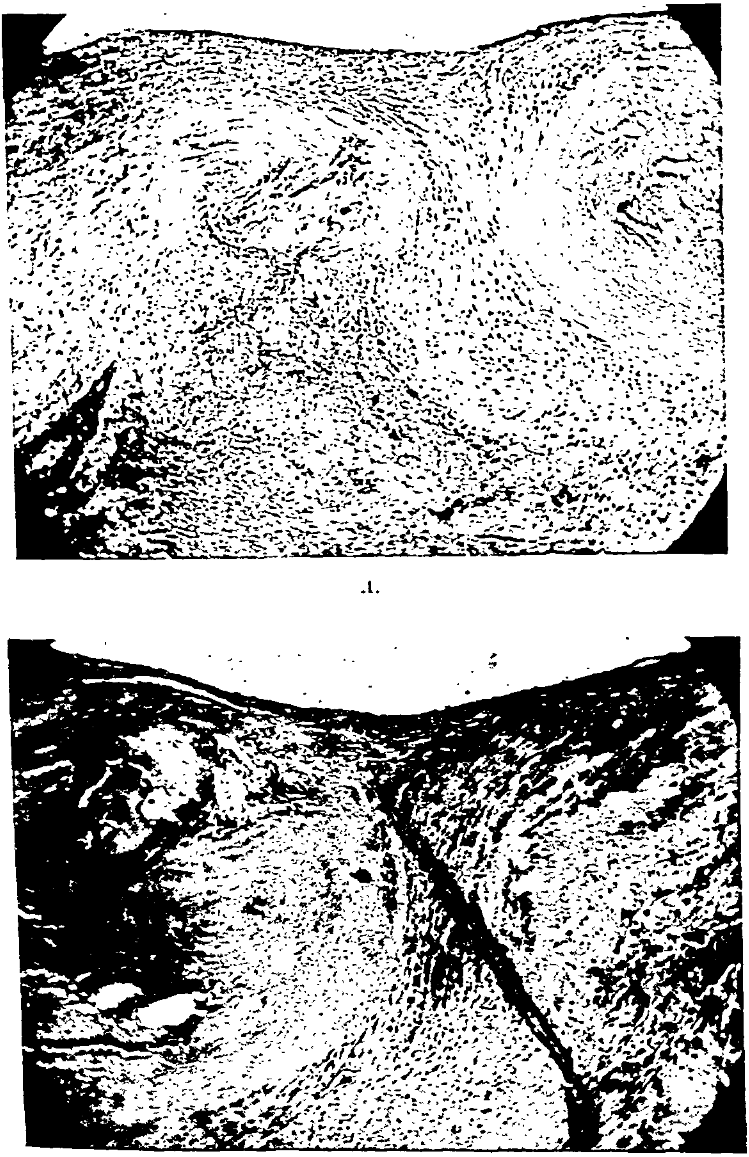


Fig. 7.—Photomicrographs of anastomoses of an abdominal aortic homograft, stored forty-five days at −27° C. and implanted for sixty-one days. (Hematoxylin and eosin. ×100.) *A*, At the silk suture line, there is excellent fibrous union and endothelization. The silk strands are unaltered, and there is little or no cellular activity about them. *B*, A fragment of chromic catgut remains in the graft at the left. Macrophages are engaged in removing this material. Excellent fibrous union and complete endothelization has occurred.

## CASE REPORTS

Case 1.—T. E., a white 21-month-old male infant, was admitted to Babies Hospital on Nov. 13, 1950, for special studies and possible operative repair of a coarctation of the aorta because of a progressive severe hypertension. The family history and past medical history were of no special interest. The child had never exhibited symptoms referable to the cardiovascular system, and had been followed closely in the pediatric clinic since the age of six weeks, at which time no cardiac murmur had been noted. However, at ten weeks, a questionable systolic murmur was noted. By the age of three months, the murmur was localized over the precordium, maximal in the left second and third interspaces. At this time, femoral pulses were not obtainable and the brachial blood pressure was 160-130/110. Laboratory studies, including chest film, electrocardiogram, Mazzini, blood culture, tuberculin, urinalysis, and fundus examination, were negative. The nonprotein nitrogen was 34 mg. per cent.

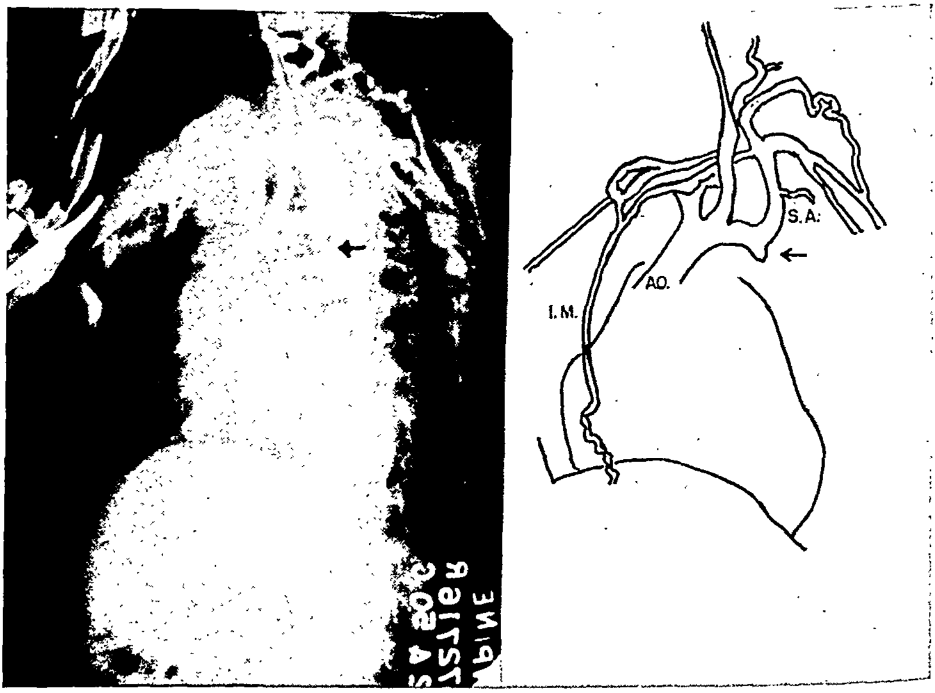


Fig. 8.—Retrograde carotid aortogram obtained in the right posterior oblique position. The obstruction in the aorta (*AO.*) is situated just distal to the left subclavian artery (*S.A.*) as shown by the arrow. Note the dilated and tortuous internal mammary artery (*I.M.*) and other collateral arteries in this 21-month-old infant.

Brachial blood pressures were obtained at six-month intervals and revealed a progressive rise to the time of admission at which time it was 180/120. Physical examination was negative except for the loud precordial systolic murmur transmitted posteriorly and the absent femoral pulses.

Positive laboratory studies carried out in November, 1950, were limited to the cardiovascular system. Angiocardiogram and a retrograde carotid arteriogram revealed a complete stenosis of the aorta about 8 millimeters distal to the left subclavian artery with minimal collateral circulation (Fig. 8). No notching of the ribs was present. Through the cooperation of Dr. John Nickerson, a recording of the low frequency, critically damped ballistocardiogram of the patient was obtained under avertin anesthesia. The initial tracing confirmed the presence of a marked coarctation (Fig. 9, *A*).

On Dec. 8, 1950, operation was performed by one of us (R.A.D.). The coarctation was of the adult type, situated as visualized at about 8 mm. distal to the slightly dilated left subclavian artery. A complete stenosis was at the site previously demonstrated. However, a very atretic cone-shaped segment of aorta constituted the 2 cm. of vessel distal to the coarctation. The left internal mammary and upper intercostal arteries were only slightly dilated and had minimal tortuosity, unlike those in our previous cases, the youngest of whom was nine years old. The ductus arteriosus was obliterated.

Although a fresh human iliac homograft was on hand, it was felt that every effort should be made to effect an end-to-end anastomosis. Accordingly, the upper four sets of intercostal arteries were sectioned and the distal aorta mobilized in its bed to below the level of the sixth intercostal arteries. In this manner it was possible to carry out a satisfactory anastomosis without undue tension after excision of the atretic length of aorta (Fig. 10). A single row of interrupted horizontal mattress sutures of 00000 medium chromic catgut was used to approximate the cut edges. An additional row of simple interrupted catgut sutures were placed in the everted edges (Fig. 11). No constriction of any significance was present at the anastomosis.

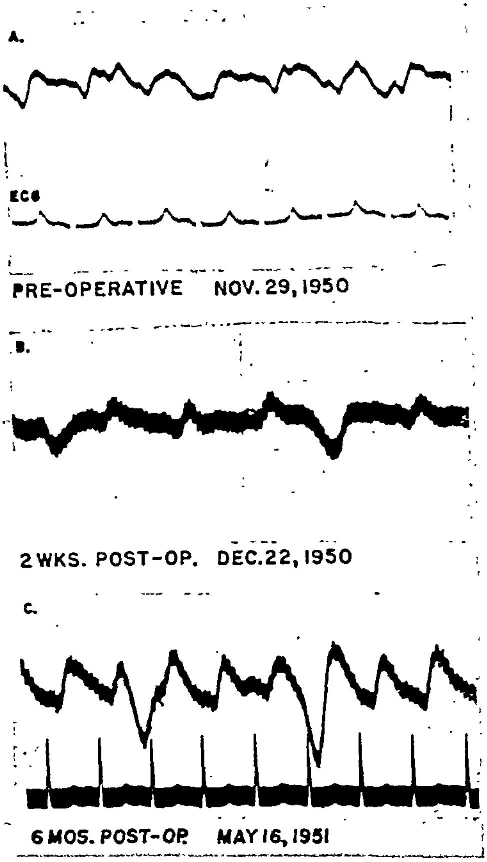


Fig. 9.—Ballistocardiograms obtained on the Nickerson bed with the infant anesthetized by aver in. *A*, Before operation, the J waves form a plateau in some complexes with a slow decline to the base line. The complete absence of K waves is characteristic of coarctation of the aorta. *B*, Two weeks after excision of the coarctation there is a more rapid down slope of the J waves with a trend toward K waves. *C*, Six months after operation, continued changes toward normal are evidenced by sharper J waves and fall below the base line. In some complexes, there are definite small K waves.

We are indebted to Drs. Hugh Fitzpatrick and Sanford Weissman for obtaining continuous determinations of the pressures in the brachial and femoral arteries by means of

Lilly manometers, during operation, and on subsequent occasions. An electrocardiogram was recorded simultaneously. Early in the operation, with the patient under ether anesthesia, the brachial blood pressure was 130/80, whereas that in the femoral artery was 85/75 (Fig. 12). Immediately before repairing the coarctation, the pressures were 110/60 and 80/70, respectively, with a lag in the onset of the femoral pulse wave of 0.14 second. Once the aorta was repaired, and the clamps had been removed slowly, there was a decline in pressures to 90/50 in the arm, and 75/50 in the leg. However, distinct improvement in hemodynamics was reflected by the greatly increased pulse pressure in the femoral pressure,

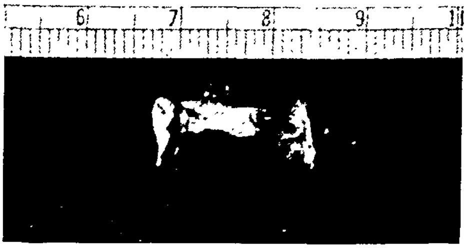


Fig. 10.—Excised coarcted segment of aorta. The proximal end faces to the left, with the ligamentum arteriosum on the superior surface at the site of the maximal stenosis.

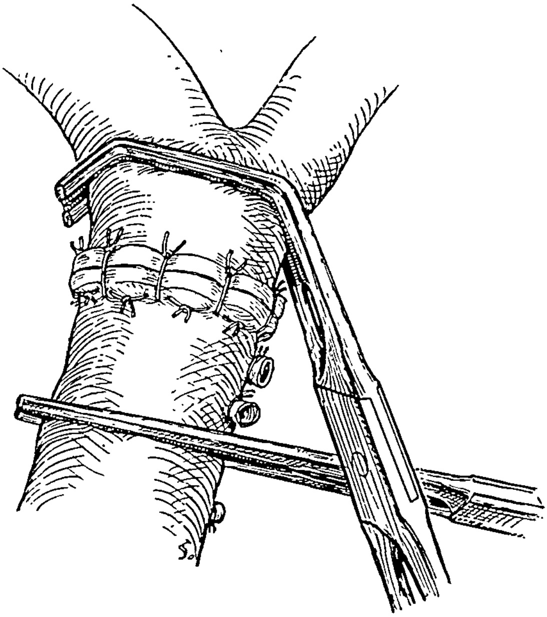


Fig. 11.—Completed anastomosis showing alternating interrupted horizontal mattress and simple sutures of 00000 chromic catgut. A straight Potts ductus clamp was used to occlude the aorta distally. The Beck clamp above permitted blood to flow into the subclavian artery during the procedure.

and in a decrease in pulse lag to 0.04 second. Two weeks later, pressures measured with the child under avertin anesthesia were 110/60 in the brachial artery and 95/50 in the femoral artery, with a pulse lag of only 0.04 second. At six months, again with the child under avertin anesthesia, these studies revealed the brachial pressure to be 120/70 and the femoral one to be 96/50. The pulse lag at this time was 0.04 second (Fig. 12).

The ballistocardiogram obtained two weeks following operation showed changes toward normal, namely, a sharper J wave and a tendency toward a K wave (Fig. 9, *B*). At six months, the J wave appeared still sharper, and a small K wave was developing (Fig. 9, *C*).

Convalescence was complicated by a slight hemothorax, corrected by a single aspiration. No tube had been employed for drainage. The patient left the hospital on the fifteenth day after operation with a brachial pressure of 120/80 as obtained by arm cuff.

Follow-up visits by the patient one and six months following date of operation revealed good weight gain. A diminishing systolic murmur was noted over the left chest. This was thought to be due to improved stream lining at the anastomosis. The pulses in the femoral and dorsalis pedis vessels were strong.

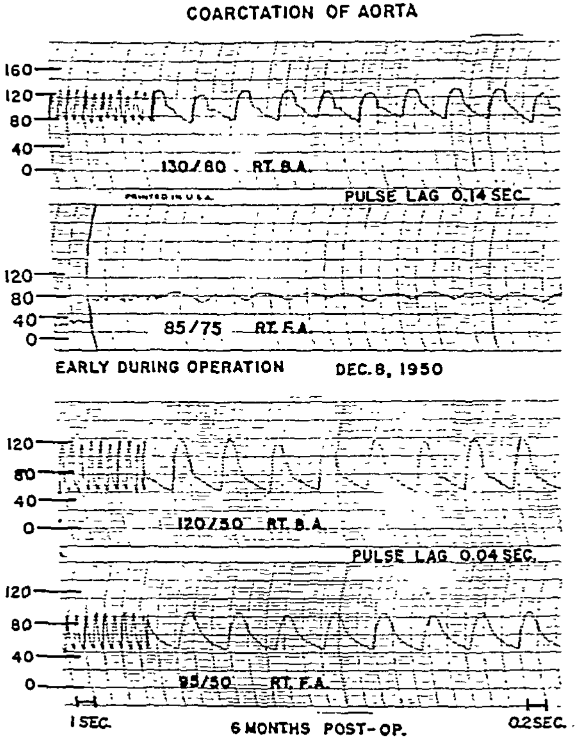


Fig. 12.—Simultaneous intra-arterial pressure tracings obtained with Lilly manometers. The upper two tracings demonstrate the pressure curves in the right brachial and right femoral arteries with the patient under ether anesthesia and before excision of the coarctation. Note the difference in onset of the waves of 0.14 second and the greatly dampened pressure in the leg. The lower two tracings obtained with the child under avertin anesthesia six months after excision of the coarctation reveal a pulse lag of only 0.04 second and greatly improved circulation in the lower extremity.

CASE 2.—W. T., a white 4-year-old boy, was admitted to Babies Hospital on May 21, 1951, with the diagnosis of portal hypertension. The child had had an omphalitis at the age of two weeks, followed at six weeks by a septic arthritis of the left shoulder. The inflammation subsided under antimicrobial therapy and his subsequent general state of health was good until the onset of fever and a head cold early in March, 1951.

Two days later, he suddenly vomited a large quantity of blood and was admitted to another hospital in shock. The only other significant information in the history recalled a fall on the abdomen a week before, with residual tenderness for several days. At time of admission March 6, 1951, his spleen was felt 2 cm. below the costal margin, but the liver was not palpable.

Blood studies revealed red count of 2.88 millions per cubic millimeter; leukocyte count, 3,400 cells per cubic millimeter, with 89 per cent polymorphonuclear cells, 10 per cent lymphocytes, and 1 per cent monocytes. The platelet count was 130,000. Bleeding time, clotting time, clot retraction, and red cell fragility were normal. Volume index was 0.93 and the icteric index, 7.4 units. Roentgenograms of the chest, skull, and long bones, urinalysis, and serological test were negative. Esophagram did reveal filling defects interpreted as varices, however.

Treatment by multiple transfusions was accompanied each time by marked splenic enlargement. The patient was discharged March 16, 1951, but had to be readmitted May 8, 1951, because of massive hematemesis. This time splenomegaly was not noted. Multiple transfusions were administered, totaling 1,500 cubic millimeters.

The local physician displayed keen clinical insight into this problem as demonstrated by his investigations and management. The child was discharged May 17, 1951, and admitted to Babies Hospital on May 21, 1951, for surgical treatment of portal hypertension, resulting from an extrahepatic block caused in all probability by the omphalitis. Physical examination this time revealed a palpable sharp liver edge, and enlarged spleen felt 10 cm. below the costal margin.

*Laboratory studies:* Hemoglobin, 9.7 Gm.; red cells, 3.12 millions per cubic millimeter; leukocytic count, 5500. Polymorphonuclear cells, 71 per cent; lymphocytes, 24 per cent; monocytes, 2 per cent, and eosinophiles, 3 per cent. Normal red cell fragility was found. Platelet count was 112,000, and reticulocytes, 2.0 per cent. Alkaline phosphatase was 2.9 Bodansky units; phosphorus, 4.5 mg. per cent; serum albumin, 4.6 Gm., and globulin, 2.2 Gm. Nonprotein nitrogen was 31 mg. per cent. Sedimentation rate 15 mm. per hour. Urinalysis negative. Chest film and excretory urogram were negative. The esophagram again showed varices. Direct Coombs test was negative. Prothrombin time was 17.1 seconds (normal fifteen seconds). Bromsulfalein retention test showed serum concentration of 29 per cent at five minutes, and 1 per cent at thirty minutes. Glucose tolerance test was normal. Blood volume determination on May 26, 1951, revealed plasma volume of 1,330 c.c., red cell mass of 550 c.c., and hematocrit of 29.1 per cent. After two transfusions of 150 c.c. each, the blood volume increased to a plasma volume of 1,350, red cell mass of 710 c.c., and hematocrit of 34.5 per cent.

On June 6, 1951, operation was performed by one of us (R.A.D.). Through a left thoracoabdominal approach, the large spleen was exposed, and the splenic vessels isolated. The splenic artery was divided and pressure exerted on the spleen to express the red cells into the general circulation. The dilated splenic vein was then divided at the bifurcation at the hilus. A springy rubber-shod Bulldog serrafin was used to occlude the proximal end until it was time to perform the anastomosis. The pressure in the splenic vein was 370 mm. of water. A large left renal vein was mobilized in the renal pedicle, and a small curved spring clamp with fine teeth* was used to isolate a 1.5 cm. segment, on the anterosuperior surface of the vein. With renal venous outflow thus unimpeded, a 1 cm. longitudinal incision was made in the segment, and the end of the splenic vein was brought into approximation. An everting anastomosis was accomplished with the use of six interrupted mattress sutures of chromic catgut. When the shunt was opened, free blood flow occurred from the splenic vein into the renal vein. Digital closure of the shunt caused a prompt rise in portal pressure of 100 to 120 millimeters of water to the previous level of 370 (Fig. 13). The portal vein was not accessible to examination, but the liver had a normal appearance. Biopsy of the liver revealed a mild cirrhosis, however.

A fine polyethylene catheter, introduced through the inferior mesenteric vein to the splenic vein, was brought out through the abdominal incision for regional heparinization of the portal blood passing through the shunt. Postoperatively, heparin in a dosage of 25 mg. per 500 c.c. of 0.85 per cent saline was administered through the catheter at a rate of 10 to 45 drops per minute for six days.

Convalescence was completely uneventful. A follow-up visit six weeks after operation revealed that the child was very active and had gained several pounds in body weight since discharge from the hospital on the fifteenth postoperative day.

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*Manufactured by Bruno Richter, *Glen Ellyn, Ill.*

CASE 3.—J. B., a white 5½-year-old girl, was admitted to Babies Hospital July 11, 1951, for special studies and possible operative repair of a coarctation of the aorta. She had been referred by her local physician to the Congenital Heart Clinic in April, 1951. Previous studies had revealed the characteristic physical signs of coarctation. Her brachial blood pressure had been noted as being 200/100. The chest film had been unusual in that there was moderate cardiac enlargement. For this reason, it was felt wise to plan an early admission.

In the hospital, a brachial hypertension of 190/96 was noted. The femoral pulses were weak and no dorsalis pedis pulses were felt. Oscillometric readings of one unit were obtained at the calf. A short harsh systolic murmur was heard over the base of the heart, transmitted posteriorly high on the left side of the chest.

Routine laboratory studies of the blood were normal, as was urinalysis. The roentgenogram of the chest and esophagram revealed enlargement of the left auricle and ventricle. An angiocardiogram on July 17, 1951, demonstrated a marked coarctation one centimeter distal to a dilated left subclavian artery. The aortic arch was shown to be very hypoplastic beyond the innominate artery to the level of the coarctation. There was a moderate poststenotic dilatation of the aorta. Difficult to interpret was the retention of diodrast in the left auricle which suggested impaired outflow from this chamber (Fig. 14). The ballistocardiogram revealed the absence of a K wave, characteristic of coarctation (Fig. 15).

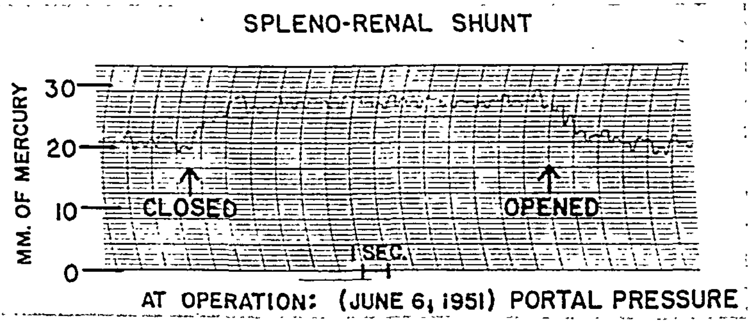

Fig. 13.—Portal pressure obtained through a catheter introduced into the inferior mesenteric vein. When the spleno-renal shunt was occluded, there was an immediate rise in pressure in the portal system. A prompt fall occurred when the shunt was again opened.

On July 20, 1951, operation was carried out by one of us (R.A.D.), after digitalization of the patient. The stenotic segment was short and situated as visualized. The aortic arch appeared hypoplastic, whereas there was a slight poststenotic dilatation below the coarctation. The fibrous ligamentum arteriosum was divided, as were the first two sets of intercostal arteries. A one centimeter segment of aorta at the coarctation was excised and a very satisfactory end-to-end repair effected as in Case 1 (Fig. 11). Examination of the specimen revealed the stenosis to be complete.

The pericardium was opened and the left auricle was examined. It did not appear to be greatly dilated, but some turbulence was noted. A systolic thrill was felt over the posterior aspect of the left ventricle. Even minimal manipulation of the heart produced arrhythmia despite topical procaine, so further investigation was thought best deferred until cardiac catheterization could be carried out.

Postoperatively, the femoral and dorsalis pedis pulses were very strong. The brachial blood pressure fluctuated between 116/80 and 160/110 during the first two weeks, as is often observed. A few days following operation the patient began to have diffuse low abdominal discomfort. Moderate fever and a leukocytosis developed, with a slowly rising polymorphonuclear count. The pain varied in intensity, but did not subside during a week of observation, and finally on July 31, 1951, exploration was performed by Dr. Robert Hiatt and one of us (R.A.D.). There was a small amount of clear yellow fluid in the peritoneal cavity. The small bowel was edematous and hyperemic. The marked hyperemia was noted in the colon and parietal peritoneum as well. All of the mesenteric lymph glands were very enlarged. The appendix was normal in appearance and there was no evidence of mesenteric or retroperitoneal bleeding. Following this operation, the abdominal symptoms subsided rapidly. It was not clear whether the mesenteric adenitis was primary or secondary. The marked hyperemia and the edema of the bowel were unusual, and the cause uncertain.

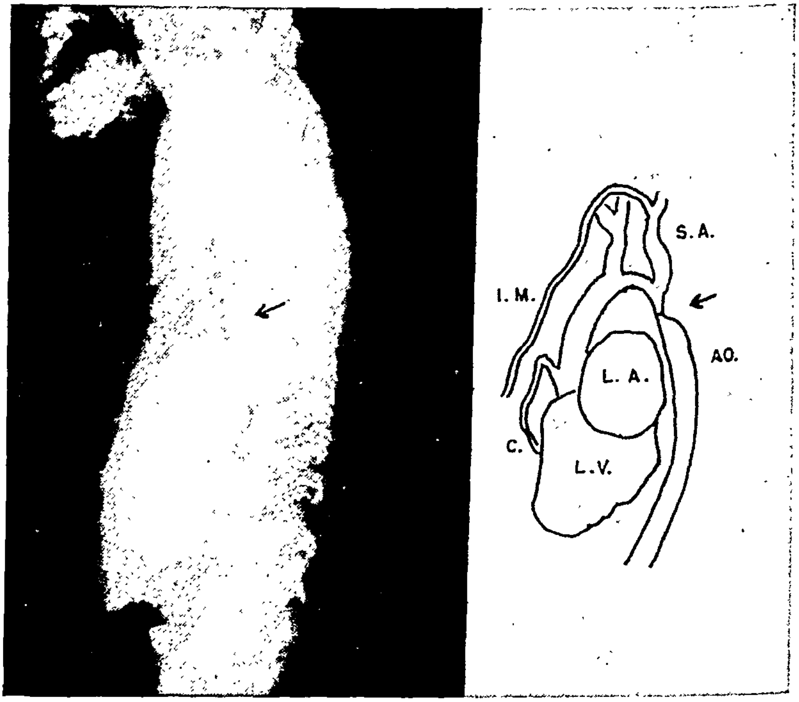

Fig. 14.—Angiocardiogram obtained of the patient in the right lateral position. The dilated left auricle (*L.A.*) is well filled with diodrast, as is the left ventricle (*L.V.*). The aortic arch is moderately narrowed beyond the innominate artery to the level of the actual coarctation (arrow), but slightly dilated below this (*AO.*). The dilated left subclavian artery (*S.A.*) coursing caphalad from the area of the stenosis, as well as the dilated, tortuous internal mammary artery (*I.M.*) are well visualized. Note the coronary artery (*C.*) anterior to the root of the aorta.

## DISCUSSION

It has not been the purpose of this study to discredit fine braided silk as a vascular suture material. Indeed, the results here would confirm that it is highly satisfactory, at the test of time has amply shown. On the other hand, this work has confirmed certain previous observations as to the very satisfactory performance of chromic catgut, when used properly.

As pointed out by Bower and Pearce,[14] many of the earlier comparisons of catgut and silk were unfair because of differences in size of the suture, type of needle used, and because of the criteria for acceptability. Even Whipple[12] called attention to greater trauma to tissues when catgut was used because of the larger needle. He suggested also that the early inflammatory response about the catgut suture always meant low grade infection. Certainly, the 00000 and 000000 catgut swaged on the needle creates no greater trauma than do the finer sizes of silk. From the extensive studies of Howes and associates,[7-11] the consistent curves of tensile strength of catgut in tissues did not support the contention that the polymorphonuclear response was on the basis of infection. Our own tissue culture studies of segments from the region of the catgut anastomosis did not show any sign of baterial infection or contamination. Jenkins and Dunham[27] have demonstrated the ability of the irritating water-insoluble hydrocarbons from the tubing fluids of both boilable and nonboilable catgut to elicit a strong polymorphonuclear reaction even when present in great dilution.

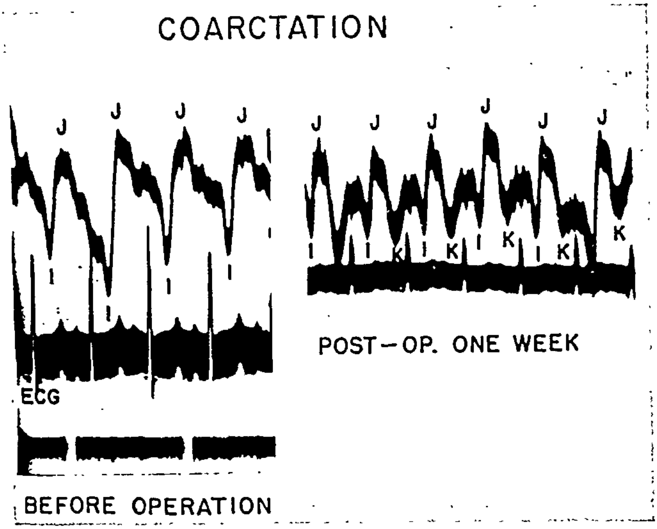

Fig. 15.—Ballistocardiogram before operation reveals the absence of a K wave which is characteristic of coarctation of the aorta. The record obtained one week after resection of the coarctation shows well-developed K waves.

They suggest that the routine technique employed in many operating rooms in preparing sutures for use is often inadequate to remove much of this fluid which is in itself adequate to explain much of the early tissue irritation. The manufacturer emphasizes the fact that precise and proper preparation of catgut, whether boilable or nonboilable, is essential in the operating room to insure ease of handling and uniformity of strength. Soaking too long in water may markedly reduce tensile strength. We admit that these factors may have been at fault at times in our own handling of this material.

The experiments by Howes[7-11] and his associates carefully analyzed the physical characteristics of catgut in various tissues, and compared this material with equivalent sizes of silk under certain circumstances. They noted a more intense inflammatory response with chromicized catgut than with plain, and showed that the time for complete loss of strength of both types of catgut approximated the time of absorption. Hence, for "20 day" chromic catgut, the complete loss of strength occurred on the eighteenth to twenty-first days. Once absorbed, no tissue reaction remained and scar formation was minimal. They observed a polymorphonuclear response with silk within forty-eight hours, as did we. Giant cells of the foreign body type were observed from the sixth day, being present thereafter as long as the silk remained. In addition, we found, not infrequently, a granuloma developing about silk strands (Fig. 5). While the petrolatum used to lubricate the silk at time of operation was considered as a possible cause for the granulomatous reaction, this has been observed with untreated silk sutures. In this regard, both Large[28] and Rosenberg and associates[29, 30] have reported on granulomas caused by the waxes used by the manufacturer to produce a frictionless finish on cotton and silk sutures.

Jenkins and co-workers[31-34] recently carried out an extensive analysis of several sizes of plain and chromic catgut marketed by ten different manufacturers. In a follow-up of over three years, they noted variability in the performance in the tissues of comparable gut of different companies. This was believed due in part to differences in treatment of the raw catgut at the factory. As a result they suggested the use of pepsin digestion tests for more accurate classification. Although the term "20 day" has been abandoned, such terms as "Type C" or "medium chromic" are being used to describe catgut such as was used in our experiments. The studies of Jenkins and associates showed such material to retain its tensile strength for ten to fifteen days, and to remain in the tissues for from three to six months, as confirmed by our observations. They noted more reaction to plain than chromicized catgut, in contrast to the findings of Howes and associates. This finding by Jenkins was later thought by him to be related to the irritation from the tubing fluid rather than to the type of gut used.[27]

Fine sizes of catgut were found most desirable by Howes and Jenkins, since the inflammatory reaction appeared to be less with the finer strands. The holding power of the tissues was found to be less in most instances than that of a 000 catgut suture line. The exception to this was found in fascia, a very condensed connective tissue. This naturally raised the question as to the strength of catgut as an arterial suture. Dry 00000 medium chromic catgut has been said to have a tensile strength of 560 grams.[18] Anastomoses made with this material were found by Sako and associates[18] to leak at pressures below those observed with silk. It should be noted, however, that the values obtained by them on the various days postoperatively were from single animals, and, consequently, these data are not fully conclusive. The delayed fatal hemorrhage observed by these authors with the everting technique and catgut is at variance with the success enjoyed by us and others.[19, 20] Their similar difficulties with the everting technique and silk suggest that the type of suture material was not a primary factor.

Howes and co-workers demonstrated that infection, hemorrhage, and tissue necrosis increased the early inflammatory reaction which hastened absorption, and hence caused a rapid loss of tensile strength regardless of chromicizing or size of the suture. In the field of vascular surgery, these are the same factors which have predisposed to hemorrhage and thrombosis regardless of suture material. There would seem to be little excuse for abandoning certain strict rules of vascular suture technique which demand avoidance of contamination, poor hemostasis, and careless handling of tissues. Certain vascular surgeons have expressed a fear to use catgut because of possible empyema, and others a desire to reinforce the catgut line with interrupted silk sutures.

In animals the use of interrupted silk sutures, when growth of the vessel is involved, has been found very satisfactory. However, the experiments carried out in baby pigs revealed that, after full growth had been obtained, anastomoses made with continuous catgut had shown a similar increase in size to those made with interrupted silk. In contrast, those made with a continuous silk suture technique showed little or no increase in diameter. Others, however, have considered significant increase in size of the anastomosis possible when a continuous silk suture is employed in a growing animal.[17, 21, 35] We noted less constriction at the catgut anastomosis after several weeks than at the silk line when considering the degree of constriction which had been present at time of operation.

The incidence of thrombus formation at the proximal suture line must be analyzed, especially since we found small mural thrombi in three instances. moderate sized in two, and complete thrombotic occlusion in two, among the twenty homografts. It is difficult to evaluate thrombus formation when homografts are used, since this incidence is no greater than that which we observed in a large series of aortic homografts implanted with silk alone.[24, 25, 26]

In our series of over seventy-five implanted aortic homografts in which 00000 arterial silk alone had been used for both suture lines, post-mortem examination has now been carried out in over half of these animals. It was noted that mural thrombosis most frequently began at the proximal suture line or in the midportion of the graft as in the group being reported here. In all instances the smaller thrombi were rapidly endothelized and became so well organized as to be identifiable only by microscopic examination.

In the carotid group it appeared that the age of the graft and inside diameter were more important factors in thrombus formation than how the grafts were stored or what suture material was used. It seems of further significance that no thrombosis was encountered in the eight carotid autografts. This superiority of the autocraft agrees with previous studies of grafts in small arteries.[36] In a further attempt to compare catgut and silk in regard to incidence of thrombosis we again refer to the studies by Sako and associates.[18] With 000000 silk used for a single end-to-end anastomosis of the aorta by dogs, there were eleven incidences of thrombi among thirty noneverted suture lines, and ten among twenty-six everted anastomoses, or an incidence of 35 to 40 per cent. With 00000 catgut, there were two instances of thrombus formation among ten noneverting anastomoses performed with chromic gut and none among three noneverting lines made with plain gut, or an incidence of 9 to 20 per cent. While statistically not suitable for quantitative comparison, there did not appear to be any greater tendency or catgut to cause thrombosis.

With regard to the clinical cases, we have usually felt that the optimum time for repairing a coarctation of the aorta is in the early teens when most of the aortic growth has been accomplished and when little sclerosis is present in the vessel. The clinical recognition of coarctation in infants and young children has not been very frequent apparently,[37-39] and reports of operation on young children and infants have been rare.[39] Our previous experience with a continuous everting horizontal mattress suture of 00000 braided silk in two children 9 years of age indicated that only partial correction of the defect may have been effected, since significant hypertension was observed subsequently. Once operation was indicated by the rapidly progressive hypertension of the 21-month-old infant, and cardiac enlargement in the 5-year-old girl, we elected to use the suture material which has appeared to cause the least late tissue reaction and scar formation, according to experimental studies.

Of the tests, the low frequency critically damped ballistocardiograph deserves mention in regard to cases of coarctation. As developed by Nickerson,[40] it differs considerably from the Starr type. The lack of overshoot allows a characteristic pattern to be demonstrated by the patient with coarctation which differs greatly from that of a normal individual.[41-45] Postoperative checks of this pattern as well as of stroke volume and cardiac output have given us valuable clues as to the effectiveness of various types of repair.

Of similar value have been the records of arterial pressures obtained directly from the brachial and femoral arteries before and after surgery. The return toward normal of the pulse lag, contour of the femoral pulse wave and arterial pressures has been charted in a number of cases by us and others.[46-49] The data obtained in our other cases of coarctation will be published in a separate report.

In general, it may be said that end-to-end repair is by far the best method of correcting a coarctation in the adult, and probably the only completely satisfactory method in the growing child. Experience in growing animals and human beings has shown that anastomosing the subclavian artery to the distal aortic stump or interposing a preserved homograft[50] may result in a partial coarctation, after full growth has occurred.

Among the cases of portal hypertension, which have been referred to us, the number of children has been surprisingly large. The usual causes have been cavernous transformation of the portal vein, thrombosis in the portal system, and schistosomiasis. Here, again, there is a distinct effort made to produce an anastomosis which can enlarge as the child grows. Whether catgut or silk is employed for anastomoses in children, one should rely on interrupted sutures as being most likely to allow maximal growth at the suture line, and certainly as the safest technique with catgut.

## SUMMARY

Fine sizes of braided silk and medium chromic catgut were used to implant vascular grafts into twenty dogs. By using both suture materials in the same animal, a comparative study was possible. The anastomoses were examined after from one to one hundred and sixty-eight days.

Grossly, the chromic catgut began to fragment in about four weeks. At times the fibrosis at the silk suture line seemed greater than that observed at the catgut anastomosis. Occlusion of some of the grafts by thrombosis was believed related to the use of homografts in these dogs, rather than to the type of suture material. Microscopically, the early cellular reaction about the catgut was greater than that about the silk in some specimens. In others, comparable but varying degrees of reaction were found about both types of suture

material. In a few weeks, excellent fibrous union occurred in all instances. Residual fragments of catgut were observed for several months. Occasionally, granuloma formation was noted about the silk strands.

The use of interrupted 00000 medium chromic catgut sutures for major vascular anastomoses is reported in three children. In two of them, aged 21 months and 5½ years, a coarctation was excised and end-to-end aortic anastomosis carried out. In the other, aged 4 years, a spleno-renal shunt was performed. In all, the clinical result has been very satisfactory to date.

We wish to thank J. A. Deknatel and Son, Inc., and Ethicon Suture Laboratories for their generous cooperation. In addition, we wish to acknowledge the assistance rendered throughout by Miss Daisy Mapes, R.N., and her assistants; the technicians in the Surgical Pathology Laboratory; and Dr. Hugh Fitzpatrick, Dr. Sanford Weissman, and Dr. John L. Nickerson.

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# Original Communications

## SIMPLE EXCISION IN THE TREATMENT OF PULMONARY TUBERCULOSIS

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THIS preliminary report concerns an experience of a year in the surgical removal of pulmonary tuberculous foci which remain after continuous daily antimicrobial therapy with streptomycin and PAS has been in force for four months or longer. The practice has been to perform local excision of diseased areas, usually with disregard for the segmental anatomy of the lung, and the rationale for this surgical procedure is based on three principles. The first principle relates to the significance of the necrotic (caseous) tuberculous focus as the cause of clinical relapses of the disease. The second principle concerns the absence of tubercle bacilli in sputum, for it is our belief that many of the postoperative tuberculous complications are related to operation performed at a time that the sputum contains organisms. The third principle relates to a conservation of good functioning lung tissue, for in most patients the most dangerous pulmonary disease is located in the superior and posterior portions of lobes of lung.

At this date, thirty patients have been operated upon, and the immediate results have been sufficiently encouraging to warrant a continuation of the program. The patients are in two distinct groups with regard to the extent of the pulmonary disease on admission to the hospital. One group consists of four patients with disease of minimal extent, none of whom had shown tubercle bacilli in repeated cultures of sputum or gastric washings during hospitalization. The logic back of the operative procedure in these cases was based upon

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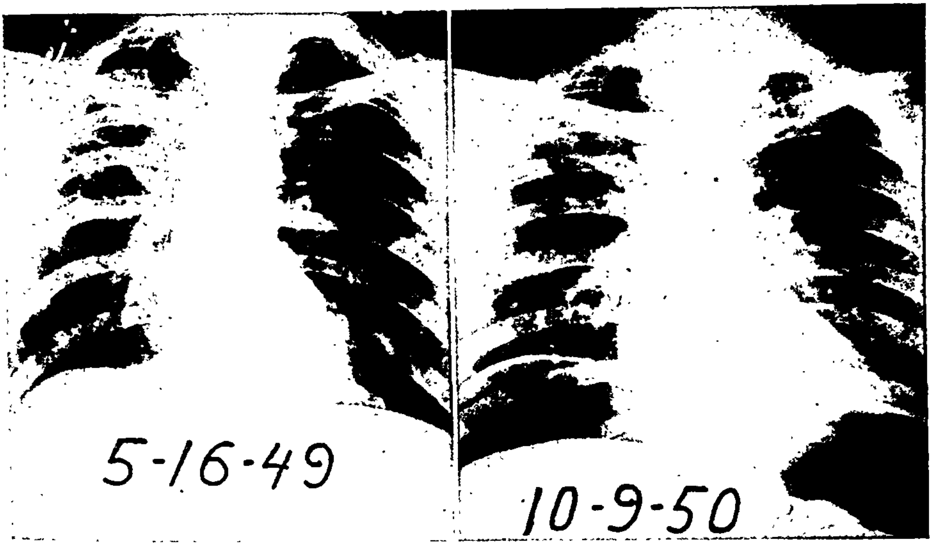
the report of one of us[1] that minimal pulmonary tuberculous disease always contains one or more necrotic lesions; and generally is a localized lesion in the superior and posterior portions of lobes of lung. It should, therefore, be possible to remove the necrotic lesions completely by a small local excision. The remaining twenty-six patients had moderately (sixteen cases) or far-advanced (ten cases) pulmonary disease on admission to hospital. None had had positive cultures of sputum or gastric contents for many weeks or months prior to surgery, although most had shown repeated positive cultures or smears of sputum prior to and early in the course of chemotherapy.

## CARDINAL POINTS IN THE PATHOGENESIS OF PULMONARY TUBERCULOSIS

Pulmonary tuberculosis is an infectious disease which is induced by the inhalation of an extremely minute air-borne particle that contains tubercle bacilli. This particle may, by chance, lodge in any portion of the lung parenchyma, and a minute microscopic focus of lobular pneumonia is produced at the site of deposit. Unless this particle lodges in a vulnerable spot in the lung (the upper and posterior portions of pulmonary lobes in the adult[1]), the diseased area seldom becomes more than a few millimeters in diameter and eventually heals without any further dissemination. The common finding of single fibrocalcific nodules in the lower and anterior portions of the pulmonary lobes at necropsy on persons who die from causes other than tuberculosis is ample proof that tuberculous foci in these regions very commonly heal. However, it is common, although not universal, for bacilli-containing particles that lodge in the superior and posterior portions of pulmonary lobes to induce a progressive pulmonary disease. *The cardinal difference between the initial disease in these locations is that the necrotic focus in the lesion in the upper portions of lobes commonly sloughs into its connecting bronchus whereas sloughing of lesions in other areas seldom occurs.* From this, it is readily seen that the process of necrosis and of sloughing of the necrotic lesion into the bronchial tree is a most important element in progressive pulmonary tuberculosis.

The complex morphologic picture that occurs as a result of the dissemination of the disease through the bronchial tree has been presented previously by one of us,[2] and at present only two phenomena will be mentioned. Frequently there are considerable areas of pneumonia which do not procede to necrosis. These areas of disease are reversible, and heal either by complete resolution or by scar formation. Other areas undergo necrosis and are irreversible. These form the well-known necrotic pneumonic lesion (the caseous lesion) which essentially is a tuberculous abscess. These absceses may soften up and slough into the connecting bronchus early or they may remain with the dead framework of the lung intact within the necrotic zone for many months or even years. All of the necrotic pneumonic areas have a tendency to slough and the larger the lesion the greater is the hazard because an organization by fibrosis is extremely slow and difficult. *A discharge of the softened necrotic debris into bronchi leads to the formation of the tuberculous cavity* and, since the necrotic area is seldom cleanly sloughed, the cavity "wall" is composed to a



*A.* *B.*

Fig. 1*A*.—A 32-year-old, white man who had acute onset of pulmonary tuberculosis in February, 1949, with progression on strict bed rest. There is a large cavity with a fluid level in the apex of the RLL, pneumonic infiltrate and multiple cavities in the RUL, and an infiltrate in the left midlung field.

Fig. 1*B*.—This is the same patient after seventeen months of continuous daily streptomycin and PAS. The pneumonic component has resolved. The cavities are filled in, leaving solid lesions.

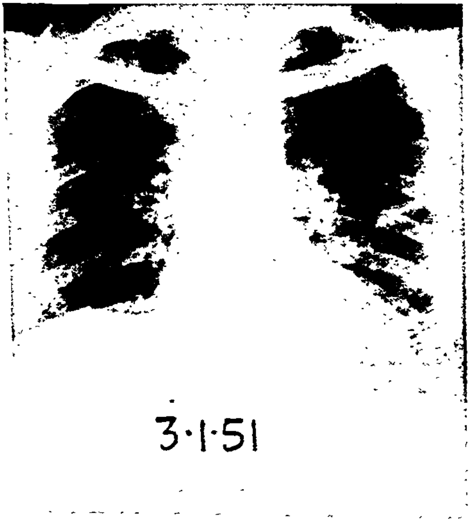


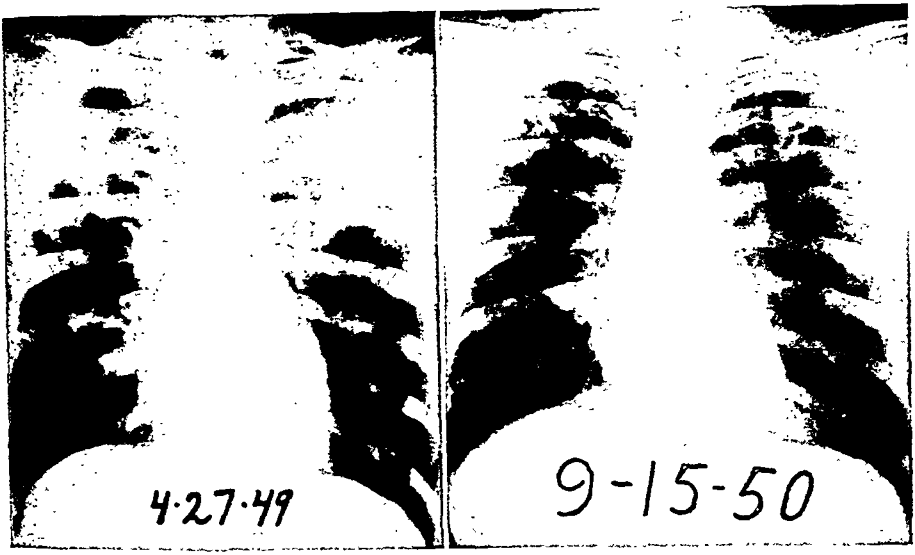
Fig. 1*C*.—Bilateral resection has been completed. Three segments and two wedges were removed from the right lung. A single wedge was removed from the lingula. He has had fourteen negative postoperative sputum cultures. M. B. C. preoperative 125 l/min, postoperative 122 l/min.

greater or lesser extent of necrotic lung tissue. It is this situation that makes a complete healing of cavity by scar formation so difficult and that leaves on "closure" of cavity an area of necrotic material surrounded by scar tissue with the patent connecting bronchus remaining or only plugged by inspissated debris. Such a filled-in cavity can again become an open cavity if the inspissated contents become sufficiently fluid to flow down the bronchus. It is the common persistence of the unhealed necrotic pneumonic tuberculous lesions in persons who have become clinically well of the disease that are the cause of relapses of the disease at a later date. These necrotic lesions usually harbor living bacilli for a long time and, although the numbers of bacilli are extremely variable, they sometimes are present in tremendous numbers even if they are not demonstrable in sputum. Unless these necrotic lesions are eradicated by one means or other, they constitute a major threat to the continued health of the tuberculous patient.

## ANTIMICROBIAL THERAPY

The effect of combined streptomycin and PAS therapy in patients with moderately or far-advanced pulmonary tuberculosis at Sunmount are being reported by D'Esopo and Raleigh.[3] The emergence of a bacillary population that is resistant to either chemical agent is greatly delayed, and the degree of resistance that develops is of a lesser order than if either agent is used alone. Clinical relapse even under continuous treatment for two years has been rare, and over 80 per cent of the patients have had their sputum become and stay negative for bacilli. A gradual and progressive clearing of pulmonary roentgenographic shadows takes place over a period of months to a point beyond which stable roentgenographic shadows remain. Tuberculous cavity-formations usually are lost to view with shadows of demonstrable size remaining. These roentgenographic changes, interpreted on a pathologic basis, are considered to represent a disappearance of the reversible pneumonic areas, a persistence of the necrotic pneumonic lesions and a filling in of cavities. These observations apply to patients who previously had not been treated chemotherapeutically. The results obtained in the retreatment of patients is much less satisfactory.

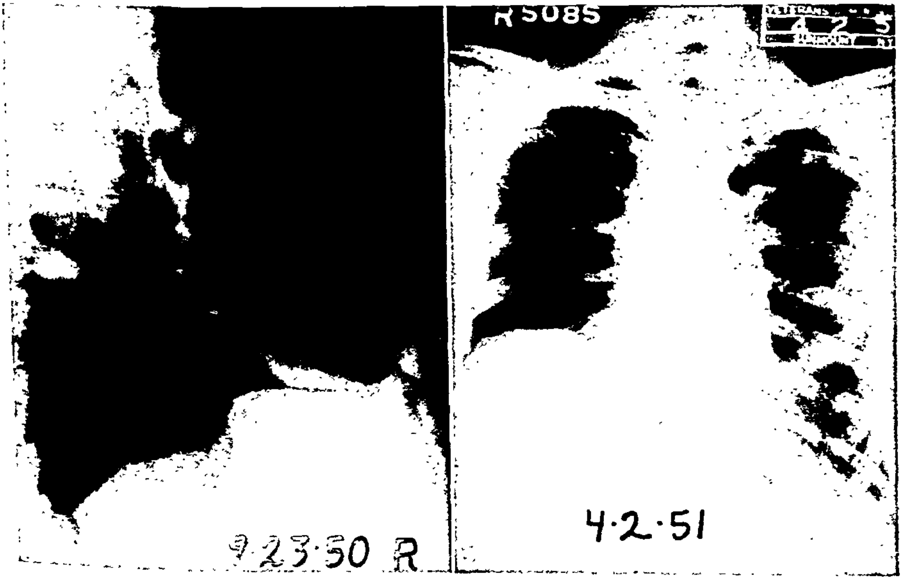
In this series, the effect of long-term streptomycin and PAS on minimal lesions has been observed both from a clinical and pathologic point of view. In the past, dissatisfaction has often arisen with the treatment of small lesions in young medical students, doctors, and nurses who have broken down during training and are so commonly sent to one of the sanitoria in the Saranac Lake area for treatment. Most of these young people have recovered permanently after a reasonable course of bed rest. However, a fair proportion of them have had second, third, or fourth breakdowns shortly after they were allowed to resume activity and, in spite of a variety of forms of treatment, have continued to be patients over a period of five or six years. It would seem logical that by doing small resections on these early localized lesions, one might eliminate, then and there, the dangerous portion of the infection and prevent these spreads and prolonged periods of invalidism. Residual necrotic foci have been



A. B.

Fig. 2A.—A 31-year-old white man who had an acute onset of pulmonary tuberculosis in February, 1949, with high fever, toxemia, and acute tuberculous laryngitis. There is a large cavity in the apical segment of the right lower lobe, a pneumonic infiltrate, and multiple cavities in both upper lobes. Streptomycin and PAS were begun at this time.

Fig. 2B.—This is the same patient after seventeen months of continuous daily streptomycin and PAS. Only dense shadows remain, representing necrotic foci and filled-in cavities. The pneumonic component has disappeared.



C. D.

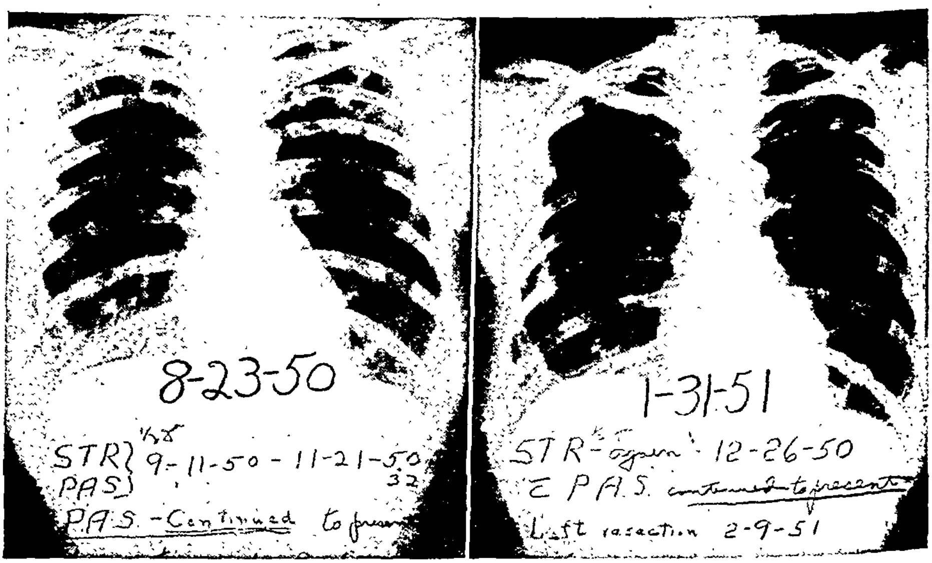
Fig. 2C.—The right lateral view shows the filled-in cavity in the apical segment of the right lower lobe and the dense shadows in the posterior segment of the right upper lobe where there previously were multiple cavities.

Fig. 2D.—Bilateral resection has been completed. Three segments and one wedge were removed on the right. On the left, three large wedges were removed. The right phrenic was inadvertently divided at operation. He has had ten negative sputum cultures postoperatively.

found in each patient operated upon for a minimal lesion, and tubercle bacilli have been shown to be present in each lesion despite repeated negative gastric and sputum cultures.

## SURGICAL APPROACH

Surgical intervention is timed to the individual patient. Disappearance of bacilli in sputum and the establishment of stationary roentgenographic picture is awaited. Advancement of the date of operation is unnecessary in the fear that resistant organisms may emerge in large numbers or that the benefit of chemotherapy may be lost before surgery is instituted. To locate and evaluate lesions prior to surgery, serial roentgenograms, lateral and oblique views, and planigrams are studied.



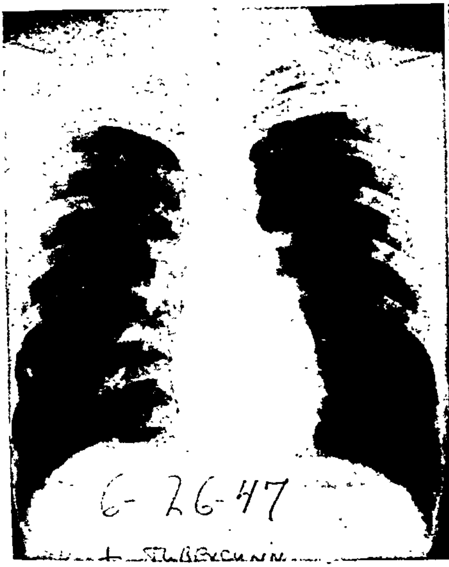
A. B.

Fig. 3A.—This is a 40-year-old nurse who contracted minimal tuberculosis in 1938. She has had four breakdowns in thirteen years and has gradually become worse. She has cavitary disease on the left side.

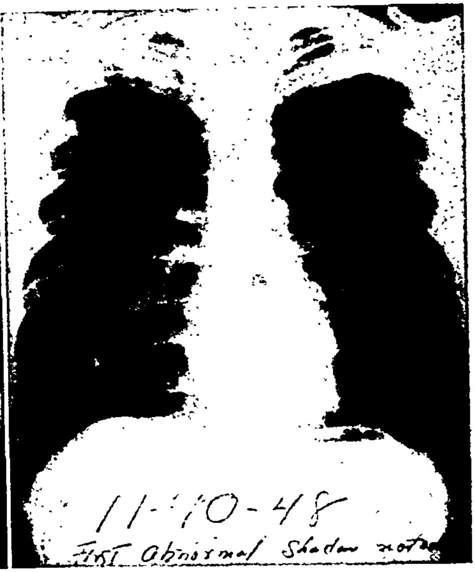
Fig. 3B.—This is following wedge resection on the right side. This is the only patient in the series in which the streptomycin course was interrupted. Wedge resection was performed on the left upper lobe on Feb. 9, 1951.

At exploration, the entire lung is carefully palpated to determine the amount of excision that is necessary. The necrotic lesions are easily detected, even if they are only a couple of millimeters in diameter. Usually filled-in cavities are easily palpated, although these may not be as firm to the touch as unsloughed necrotic foci. Commonly the lung feels normal in large areas which previously had shown extensive roentgenographic shadows. Little difficulty has been encountered in locating lesions which were indicated by roentgenographic shadows; however, it has not been unusual to find significant lesions which were not suspected from study of roentgenograms.

The amount of lung tissue to be excised is determined after a complete palpation of the lung has been made with the preservation of a maximum



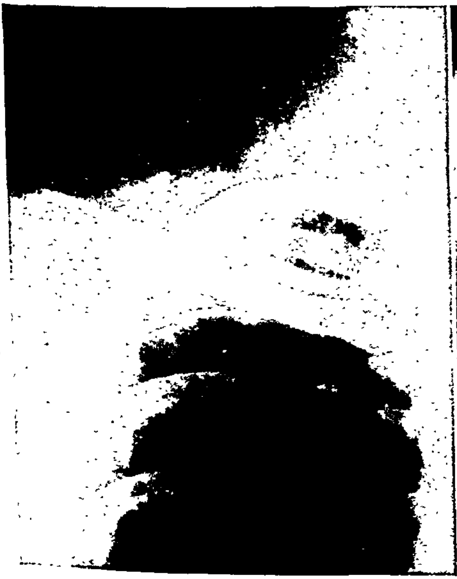
A.



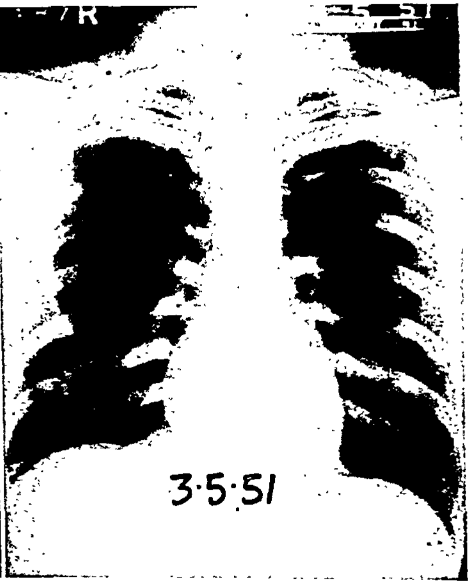
B.

Fig. 4A.—This is a 27-year-old physician whose tuberculin became positive at this time. It had been negative less than a year previously. No abnormal shadow was seen in the roentgenogram.

Fig. 4B.—An abnormal shadow was first noted at the right apex at this time.



C.



D.

Fig. 4C.—This is a magnification of the previous film showing the lesion at the right apex.

Fig. 4D.—A wedge of necrotic tuberculosis (minimal lesion) was removed from the apex of the right upper lobe. The patient received four months of streptomycin and PAS preoperatively, and four months of both drugs postoperatively. Since Jan. 1, 1952, he has been working full time as a resident physician.

amount of functional lung being carefully considered. Usually only wedges of lung are resected, although on occasion the removal of an entire lobe or a lung may be necessary. Some patients have many small nodules scattered throughout a lobe or a lung and, in such instance, one hesitates to sacrifice so much healthy lung parenchyma. The nodules located in the superior portions of lobes are resected, usually by wedge resection, because they are located in the vulnerable portions of lobes.[1] Wedges may be removed from other parts of the lung if numerous nodules are present in the area. In any location, lesions over 0.5 cm. have been removed singly. As many as nine wedges have been removed from the same patient. Minute lesions in the lower portions of lobes are left undisturbed on the pathologic basis that there is a strong tendency for small necrotic lesions in such locations to heal.

The surgical technique in itself is simple. The wedge to be resected is outlined, the pleura is incised a short distance beyond the disease area, and the ara is excised en bloc. Sutures are applied to bleeding points and bronchial leaks. Usually the large bronchus at the apex of the wedge has appeared to be normal and is easily ligated. In most instances, no effort has been made to pleuralize the raw surface or to close the defect by approximating the edges. However, in a few instances, persistent small bronchial leaks have been controlled either by placing gelfoam or pleural grafts on the raw surface. Not infrequently, the disease may appear too deeply located to allow a wedge resection. However, experience has shown that after the segmental vessels and bronchi have been dissected out, the diseased area can be lifted away from the hilum, and a wedge or subsegmental resection can be performed.

## EARLY RESULTS

Prior to Feb. 1, 1951, thirty consecutive wedge or subsegmental resections have been done with bilateral resections in three patients. The pathology in each resected specimen showed necrotic pneumonic lesions and the cavity-bearing areas removed showed filled-in cavities with patent bronchial communications. Otherwise there were areas of scarred tissue with extremely little evidence of unhealed nonnecrotic pneumonic lesions. The bronchi at the resected end of the wedge have showed little evidence of disease other than some lymphocytic infiltration of an intact mucosa. Tubercle bacilli have been demonstrated in one or more necrotic lesions in every case, although not in all lesions. Patent bronchial communications with some necrotic lesions were demonstrated microscopically, although they were not found macroscopically. Two hundred three postoperative cultures of sputum or fasting gastric contents for tubercle bacilli have all given negative results. There has been little change in the cardiorespiratory reserves postoperatively, as compared to preoperative findings. Nearly all have had preoperative and postoperative pulmonary function studies. No postoperative tuberculous complications have arisen. No complications of any sort have occurred in the four patients with minimal disease. As a rule, the postoperative course has been benign. At present, all patients are in excellent physical condition. The following is a list of complications that have been encountered and that one might expect to occur following the surgical procedure used:

| | |
|---|---|
| Number of Operations | 30 |
| Hemothorax requiring postoperative evacuation | 1 |
| "Atelectasis" requiring bronchoscopic evacuation | 1 |
| Division of phrenic nerve (inadvertent) | 1 |
| Wound infection | 0 |
| Empyema | 0 |
| Bronchopleural fistula | 0 |
| Increase of pulmonary tuberculosis | 0 |
| Total | 3 (10%) |

## CONCLUSION

It is fully recognized that these early results give only a part of the picture, and that a lapse of five years may be necessary for the story to be completed. It is not claimed that this approach to the surgical problem of the tuberculous lung will eradicate all of the pulmonary disease; however, it is anticipated that the small lesions left behind will cause no further trouble. There is maximum conservation of normal functioning pulmonary parenchyma. However, if this approach to the problem does not yield results superior to other accepted methods of treatment, then it can be definitely concluded that pulmonary tuberculosis is not a disease that can be controlled by surgical excision.

## ADDENDUM

Feb. 18, 1952. Follow-up to date reveals no clinical or roentgenologic relapse in this series. One patient has had two positive sputum cultures since operation. He had received previous courses of streptomycin.

## REFERENCES

1. Medlar, E. M.: The Pathogenesis of Minimal Pulmonary Tuberculosis, Am. Rev. Tuberc. 58: 583, 1948.
2. Medlar, E. M.: Pathogenetic Concepts of Tuberculosis, Am. J. Med. 9: 611, 1950.
3. D'Esopo, N. D., and Raleigh, J. W.: Am. Rev. Tuberc. in press.

When this paper was presented at Atlantic City the section in the text on Pathology was omitted due to time limitation. The following discussion by Dr. Welles was put in to clarify the pathologic concepts on which the surgery was based.

The slides referred to in Dr. Welles' discussion are color plate Illustrations I and II.

## DISCUSSION

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—I am sorry Dr. Medlar is not here to discuss the pathogenesis and the pathology of tuberculosis. Most of us probably feel that we understand the pathology of tuberculosis thoroughly, but I have had to change my way of thinking about it since associating with Dr. Medlar, and possibly I can explain his thoughts to you.

We have been taught in the past that the process is the invasion of the lung by tubercle bacilli, followed by acute inflammatory reaction with invasion polys which are later replaced by lymphocytes and giant cells; caseation can occur in the center of a lesion and then be replaced by the infiltration of fibroblasts and formation of a scar. The trouble with thinking about tuberculosis in that way is that that happens in microscopic lesions and the demonstration of that process which has been shown so well by many pathologists and which was taught for years by Dr. Leroy Gardner, is that the demonstration was made with a microscope. All that picture can be shown in the microscope. But Dr. Medlar has shown

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Illustration II.—This illustration shows a "necrotic" or "caseous" tuberculous pneumonia of about the same size as in Illustration I. This is the "irreversible" phase of tuberculous pneumonia. This phase of the disease remains after "bed rest," pneumothorax, and chemotherapy, and constitutes the hazard of "relapse" of the disease after "clinical" recovery. It is this type of lesion which gives rise to cavity formation if it softens up and sloughs through the bronchus which connects with the necrotic area. In this particular lesion no bacilli were found however in many lesions which are morphologically identical bacilli are often present in large numbers. This lesion is an endobronchial dissemination or metastasis and was present in the lower posterior portion of an upper lobe.

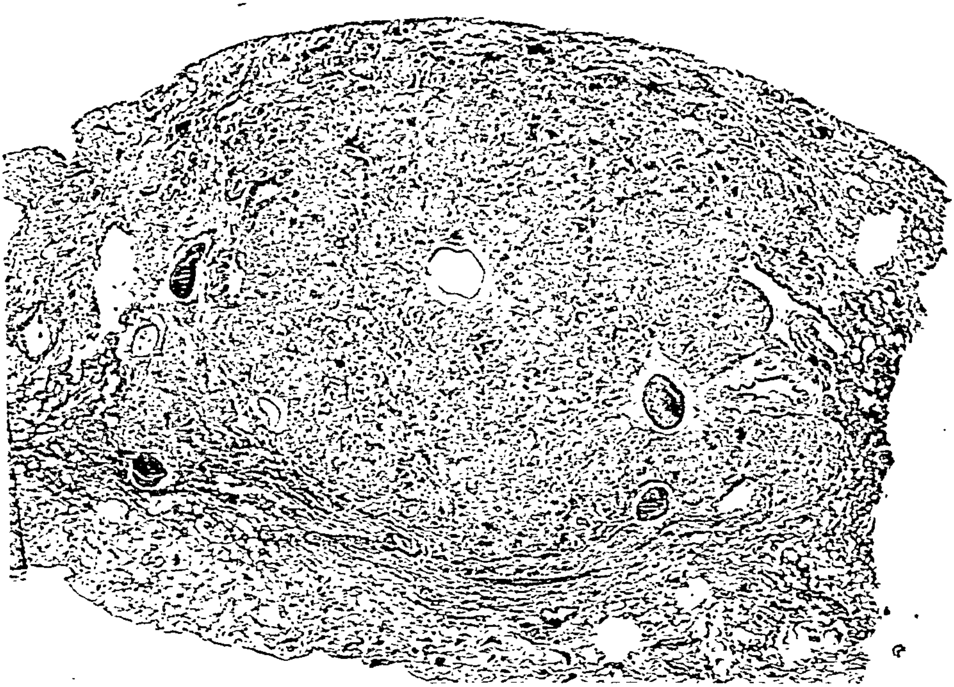
Illustration III.—This illustration is from the apical area of the same lobe as the lesion in Illustration II. There are five small "necrotic" pneumonic lesions present. One of these small lesions showed large numbers of bacilli. Since the superior portions of pulmonary lobes are the most vulnerable portions of the lung in tuberculosis, lesions of any size in these regions should be excised when palpated on exploration of a lung. Both size and location of lesions need to be considered in the decision of excision.

Illustration IV.—This old necrotic lesion was removed from a patient who had been known to have pulmonary tuberculosis for eight years, and who had had four separate hospital admissions. Before surgery the patient had been on continuous combined streptomycin and PAS for over a year. The lesion illustrated measured only three millimeters in diameter and was not visualized in roentgenograms, but was easily palpated at operation. This represents one of the small local excisions commonly made. Note the bronchus connecting directly with the necrotic area and having necrotic debris within its lumen. The bronchial lining epithelium is normal up to the point at which it abuts upon the necrotc area. A peripheral zone of well-formed fibrotic tissue surrounds the necrotic area except at the point of bronchial connection. This is a common situation and is the principal factor which allows pulmonary tuberculosis to be an unpredictable disease that has a strong tendency to relapse. Bronchi commonly are plugged by necrotic debris but rarely are obliterated by scar formation across their lumen.

Illustrations V and VI.—These two illustrations are from the two lungs of the same patient. Illustration V shows the pathology found in a "wedge" resection after combined streptomycin and PAS had been in effect for one month. Both "nonnecrotic" and "necrotic" areas of tuberculous pneumonia are present. Illustration VI shows the pathology present in a "wedge" resection from the opposite lung after combined streptomycin and PAS therapy had been in effect for six months. During this time considerable "clearing" of roentgenographic shadows had occurred in the area from which the "wedge" was removed. In this section only "necrotic" lesions and some scar formations are present. The lesion represented in Illustration V had shown a stationary roentgenographic shadow for five years. Tubercle bacilli were demonstrated in necrotic lesions on both sides, both in tissue section and by culture. Although this patient had had four relapses over a period of twelve years, tubercle bacilli had never been demonstrated in the sputum.

Illustration VII.—This illustration is a cross-section of a small wedge of lung tissue removed from a patient with "minimal" pulmonary tuberculosis. The patient first reacted to tuberculin in 1947, and showed a small shadow in the area resected in 1948. The shadow persisted after one year of "bed rest" therapy and an additional four months of combined streptomycin and PAS therapy. The wedge of lung removed measured about 2.5 cm. across. Serial section of the entire specimen showed the presence of six separate lesions—two of which were largely scar tissue and the other four were similar to the lesions in the illustration. In one of the scarred areas was an organizing old necrotic focus which was interpreted as the initial necrotic lesion from which the others were disseminated. The three lesions in the illustration show a peripheral organizing zone of granulation tissue and a central "core" composed in large part of neutrophiles. Tubercle bacilli in small numbers were found in the exudate in these lesions but were not found in the scarred areas. Only an occasional epithelioid tubercle and giant cell was found in the serial sections.

Illustration VIII.—This illustration is from a small wedge of lung tissue removed from a patient with a history similar to that of the patient illustrated in Illustration VII, with the exception that the date of conversion to a reactor to tuberculin was not known. Also this patient showed an increase of disease near the end of his course of bed-rest therapy. He was then started on combined streptomycin and PAS therapy and after five months of this treatment the roentgenographic shadows had receded to their original size. The specimen removed contained two fibronecrotic foci which measured about three millimeters across and a larger lesion which measured six tenths by one centimeter. The largest lesion was serial sectioned and the illustration from one level of the serial sections. This lesion proved to be a minute cavity formation partly filled with necrotic material and it also had a patent bronchial communication. An occasional tubercle bacillus was found in the necrotic contents of the cavity. At one side of the larger lesion is seen a fibronecrotic focus which measured less than two millimeters across and which had a small calcific center. At another level this old focus becomes an integral part of the larger lesion and it seems probable that this was the source from which the larger lesion had its origin. This clearly portrays the complexity frequently encountered even in small and seemingly insignificant tuberculous lesions.



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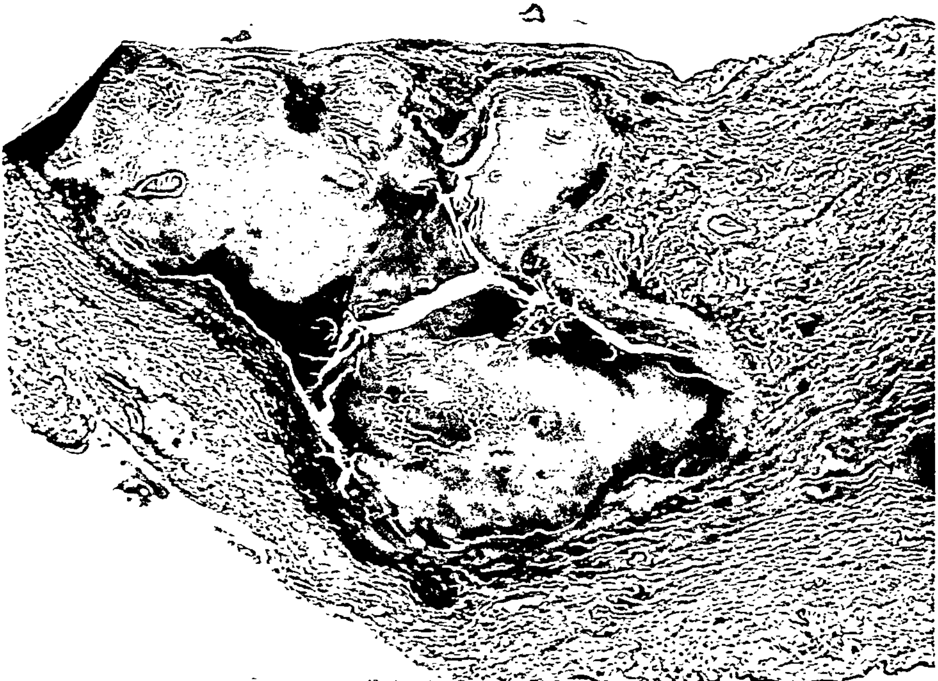

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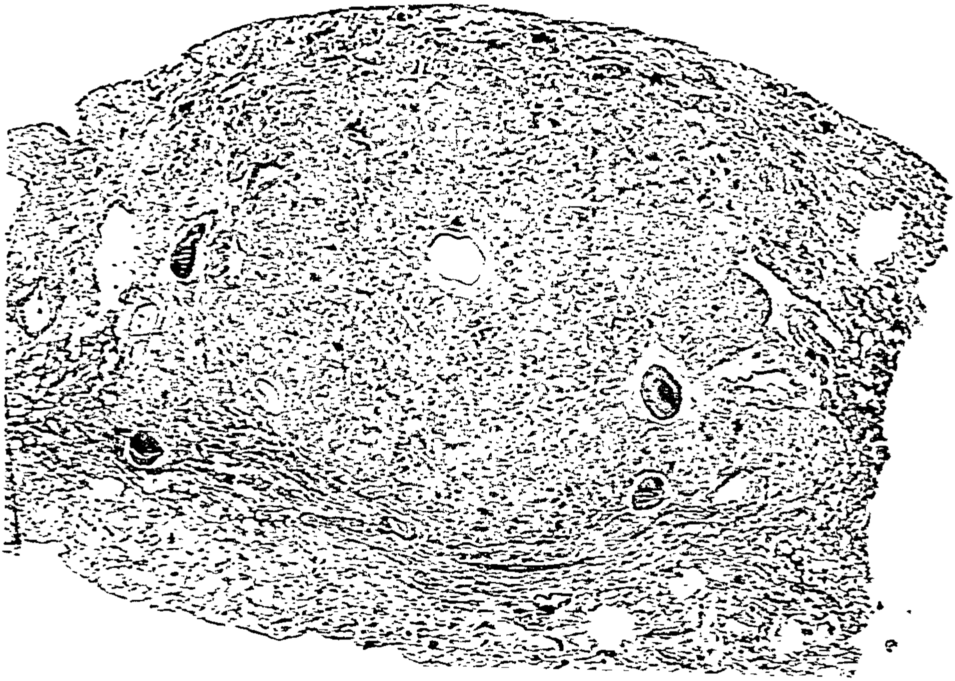

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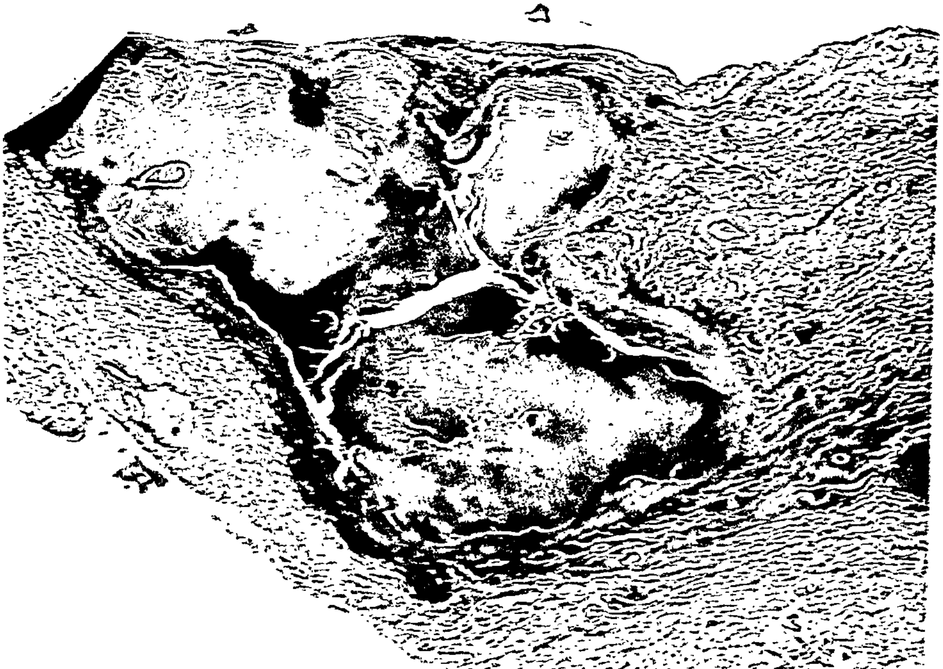

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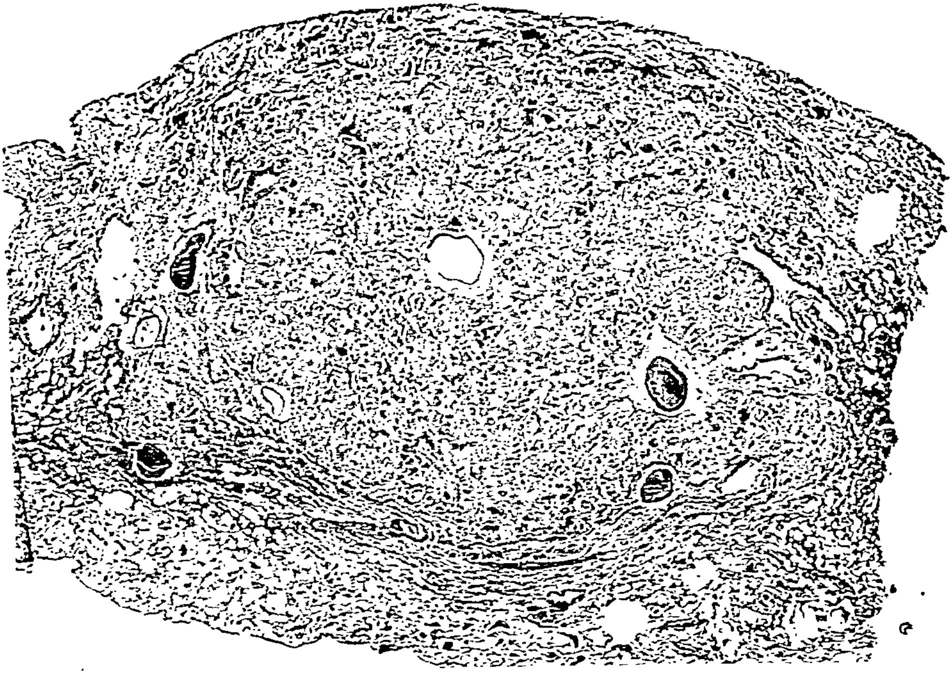

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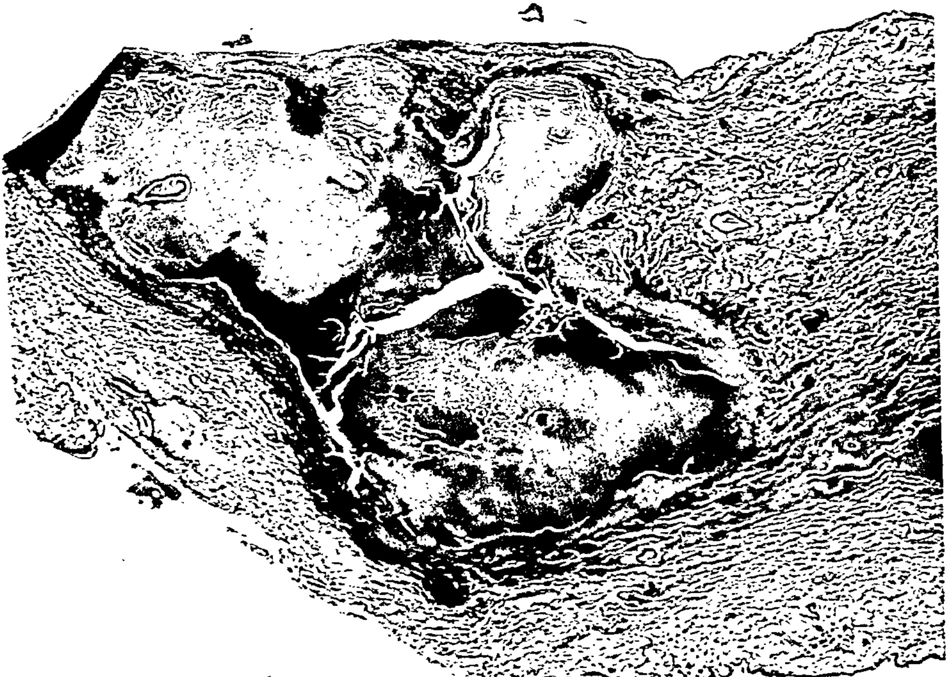

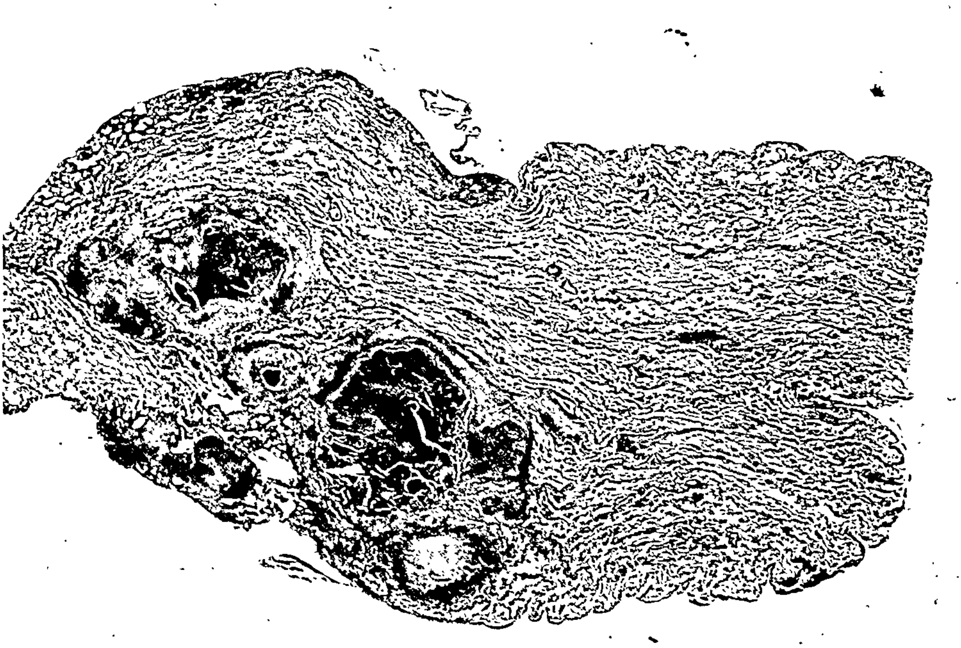

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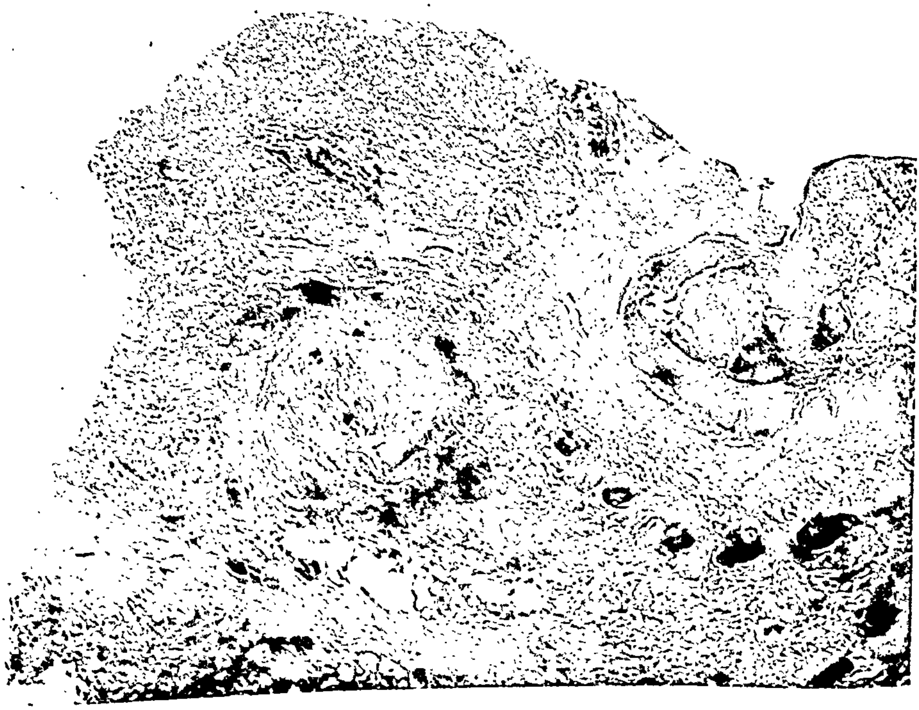

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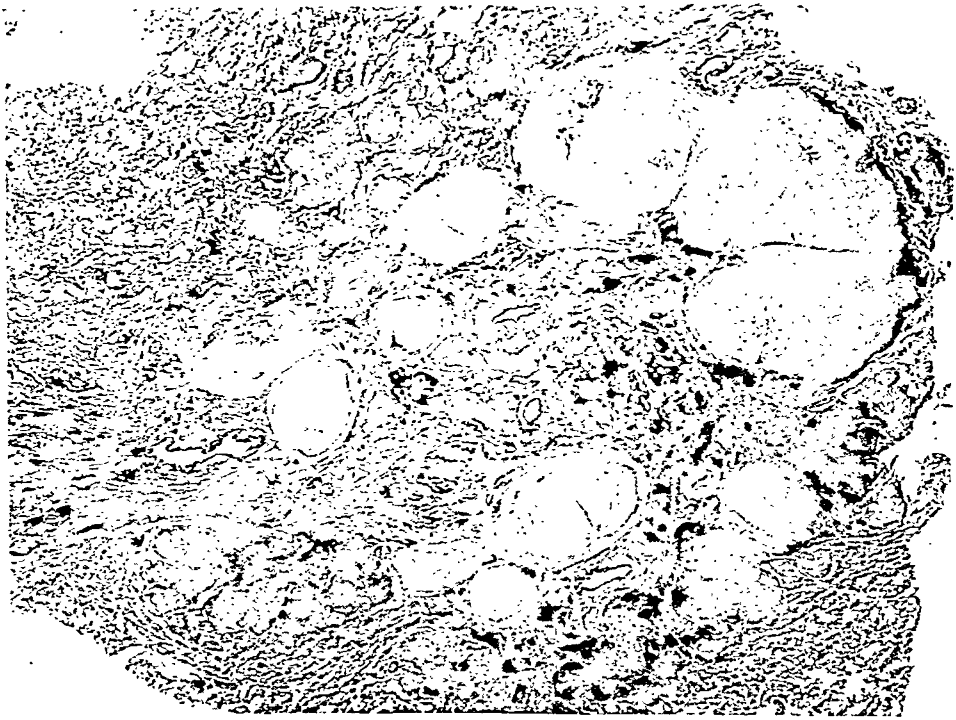

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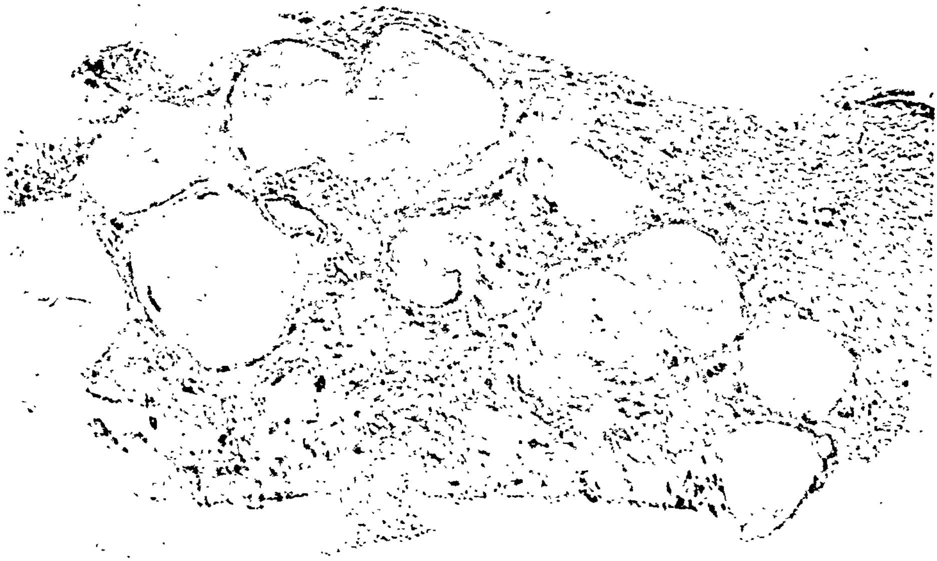

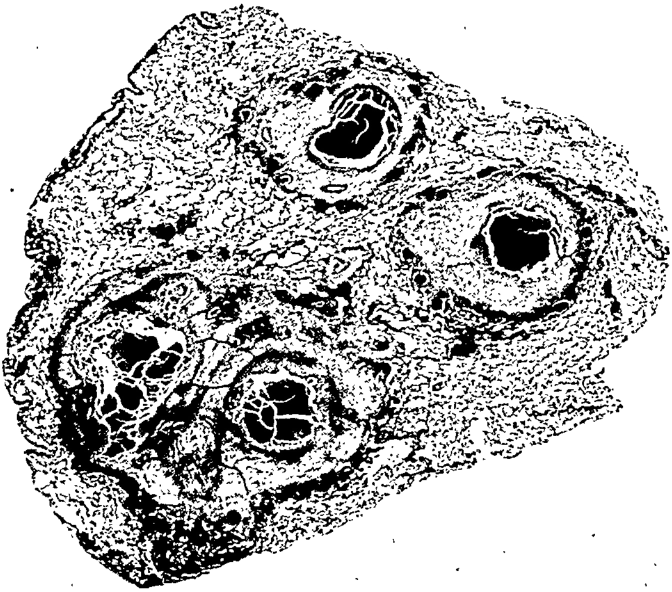

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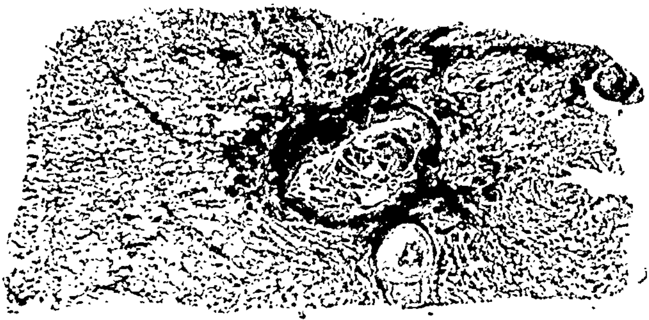

over the years that there are definitely two types of tuberculous processes in the lung. If a lobule of the lung is invaded by a relatively small number of tubercle bacilli, and their virulence is not too high, tuberculous pneumonia occurs. If another lobule is invaded by an overwhelming number of bacilli, or very virulent bacilli, the tissue is killed and necrosis occurs, and these necrotic areas can be of almost any size.

(Slide.) (Color plate I.) This is the typical lobule full of tuberculous pneumonia. There is no destruction of tissue except in small microscopic areas. You will get a better idea of the lesions Dr. Ryan showed if I tell you the size of these. None of the colored slides shown by Dr. Ryan, and neither of these, is a magnified microphotograph; they are sections. This is an actual section of the lung, and the size is about one centimeter vertically by two centimeters horizontally. This is a reversible lesion. Under rest treatment this lesion can go on and heal and leave practically normal lung. Under antibiotic treatment it will heal and disappear and leave a clear shardow in the roentgenogram. At the present time this lesion leaves a solid shadow in the film which is indistinguishable from the lesion shown in the next slide.

(Slide.) (Color plate II.) This is almost identically the same size lesion, about one centimeter by two centimeters—as large as the joint of my thumb: There is no magnification here except what is given by the projector. This is a necrotic, destroyed area that cannot return to normal under rest treatment, under pneumothorax, under antibiotic treatment. Fibroblasts cannot work into this lesion from the capsule, replace the necrotic caseous material, and turn this lesion into a scar. I have had arguments with pathologists—"We have seen this fibrosis take place in necrotic lesions." I remind them that they have seen that happen under the microscope, but have not seen it happen in lesions as big as the end of my finger like this one. The only way this lesion can disappear is by liquefying, emptying out through its bronchus, which is there and open. That can happen without spread of tuberculosis if the material is all coughed up; or it can produce spread as it is liquefied. If it liquefies, empties out, becomes then a thin-walled cavity, the walls can fall together and it can become a scar.

These are the lesions left after long-term antibiotic therapy. These are the ones we are removing and these are the ones we are finding in minimal disease and taking out, hoping that we will prevent recurrence over and over again in these young people.

(Slide.) This is a diagram that Dr. Ryan drew to show that the connection of the bronchus to the necrotic lesion is present, but the finding of it and showing it in the section is a matter of luck. We have often been told by the pathologists that a lesion is completely encapsulated. If the section is cut where the vertical line is you will find, as you saw in many of Dr. Ryan's sections, a completely round encapsulated lesion with a rather thin capsule all the way around it. Obviously it has no bronchial connection. Obviously it is safe. However, if the section of the same lesion is made in the direction of the slanting line or horizontally at the level of the bronchus, the bronchial connection is immediately apparent. As Dr. Ryan said, bronchial connections, by careful examination by the pathologist, have been demonstrated in practically all these lesions when he took the trouble to look for them.

I simply hope this will give a better picture of our conception of the pathology, when the criticism and discussion start.

DR. JEROME HEAD, Chicago.—During the past few weeks, in my spare time, I have been reading Oswald Spengler's *Decline of the West*. He gives as an inclination of declining civilization the development of virtuosity without a solid basis in reality. I mention this because I feel that as clinicians and surgeons there is a danger that we are giving way to such virtuosity; that we are turning our clinical functions over to the pathologist and bacteriologist. If the bacteriologist by the most delicate methods can culture a single tubercle bacillus from a patient's gastric washings, we are argued into believing that the patient is ill and in need of the excision of something—perhaps a segment of his lung, perhaps a whole lung. We must ask ourselves seriously if the patient with an occasional positive culture on gastric wash following thoracoplasty is a menace to his associates or a menace to himself. Is such a finding an indication for pneumonectomy? I do not believe so.

Similarly, is a patient on whom the pathologist can demonstrate a walled-off focus of tuberculosis or a minimal tuberculous infiltrate which has not healed entirely in the center, clinically tuberculous or clinically threatened? I do not believe so. I believe that we must insist on our rights as clinicians and that in these matters we must refer to experience and statistics.

At Edward Sanatorium, 98 per cent of the patients who entered with minimal tuberculosis were well five to ten years after their treatment with bed rest or pneumothorax. Our results on all the patients who are discharged as arrested show that 85 per cent are well from five to ten years after their discharge. In other words, our over-all recurrence rate is between 10 and 15 per cent. In view of these statistics, I do not feel that we must surgically eradicate every shadow that appears on the roentgenogram. What Dr. Medlar has shown and what Dr. Ryan and Dr. Welles are pointing out, is that we arrest tuberculosis, we do not cure it. After all, what we are after is people who, after treatment, are clinically well and who remain clinically well. I am not at all interested in people who are bacteriologically pure or pathologically beyond criticism.

In saying this I do not mean that this paper is not worth while. It is probable that we will find that certain very large infiltrates are so dangerous as to warrant their resection. But to proceed from this to argue that every infiltrate should be removed is, in my opinion, a tendency toward virtuosity.

DR. ROBERT G. BLOCH, Chicago.—To follow up Dr. Head's remarks, I take the *Decline of the West* for granted but not the decline of tuberculosis. One of the reasons for this is the very type of lesion which Dr. Ryan and Dr. Welles discussed. The so-called early reinfection lesion in a majority of cases proves to be pathologically further advanced than is frequently thought. Any sharply circumscribed shadow of moderate or little density, if it is proved to be tuberculous, can be assumed to be a caseous focus. As such, it is a potential cavity or, in fact, it is a cavity which as yet has not found communication with the bronchial system or where that communication is temporarily blocked. These early reinfection caseous tuberculomas remain the residual problem in tuberculosis, although as time goes by most or all of the involvement emanating from them through the years may heal satisfactorily. It is this residual tuberculoma and potential cavity which poses the essential problem. It is justified to make it one of the aims of surgical attention as the authors have done, but I agree with Dr. Head that it should not be necessary to excise all these lesions. Enough circumferential fibrosis can occur to render them clinically innocuous. If the central caseation is small enough, calcification may eventually occur to result in pathologic healing.

This discussion involves the question of what we should call active tuberculosis. Active tuberculosis is generally conceived as an involvement which is undergoing changes in either direction. This would imply that lesions which fail to do so over a period of time should be called arrested. I believe this is a fallacy and dangerous; any of these so-called arrested lesions which contain caseous material, the breeding place of organisms, is so highly potentially active that to term it as arrested and to send the patient back into active life with that classification is likely to lead to misunderstandings about the further possibilities of the course of the disease. Lesions, therefore, which assumedly are caseous tuberculomas, should be classified as "potentially active." On the other hand, the sound caution, which is always indicated in judging tuberculosis as healed, has induced the authors of the newly republished *Diagnostic Standards and Classification of Tuberculosis* to omit the terms "healed" or "cured" entirely. According to this classification, therefore, the disease is not curable. I should like to go on record as objecting strenuously to that concept, which I believe to have originated largely from the very region from which the authors of this paper hail. We must steer clear of the dangers of overclassification, and provide leeway for a variety of interpretations based on the merits of each individual case. Let us avoid standardization in all aspects of medicine. One of the discussors of the first paper this morning demanded standardization of surgical technique in tuberculosis. It was one of the most horrible words I ever heard at a medical meeting. Once we reach the point where we want standardized concepts and technique, we will have reached the end of progress.

DR. FRANK B. BERRY, New York.—I think anyone who has followed this work of Dr. Medlar, Dr. Welles and Dr. Ryan carefully, and who has seen their results and talked with them, cannot but be convinced of its soundness and correctness. The lesions you saw on the films and later the sections as shown you by Dr. Ryan, were the residua. They were demonstrable residua of caseous foci, and we all know that caseous foci break down and are a danger; they liquefy and discharge through a bronchus as Dr. Bloch has just told you. The physician whose small apical lesion you saw has come under discussion before, at meetings which I have attended. But I am sure that if it were your lesion and you knew it had not changed for more than a year, and you were walking around with a caseous focus, I am sure you would feel better without it than still carrying it with you.

DR. WARRINER WOODRUFF, Saranac Lake, N. Y.—I am on my feet because I have been following this work closely and, to my mind, this is one of the most important papers dealing with tuberculosis to which I have had the privilege of listening in this Association. I think it represents a fundamental change in concept. Many of the papers we have presented and listened to have been primarily papers on technique. There is still a place for that. Obviously, technique is a very important part of this work. When the authors can present twenty-five consecutive cases of excisional surgery in pulmonary tuberculosis, with no mortality and this low morbidity, that is excellent technique; but I think far more important than that is the change in thinking. In the past our goal has been to arrest the disease and enable the patient to live with it. Dr. Pannier, in his discussion this morning, told of the number of late thoracoplasty deaths from tuberculosis. Any of you in sanatorium work know that, in any series of admissions in any sanatorium, a considerable number are repeaters. The young doctor whom they were discussing this morning—I do not know his identity—is an example, but we have all seen patients who, at one time in the course of their disease, have had a lesion no larger than his. Many permanently arrest their disease, but others we have watched break a second, a third, or a fourth time.

If it is possible to excise the tuberculosis (I realize you cannot get rid of all the bacilli) allowing the body defenses to take over from that point; if, over a period of years it can be shown that the relapse rate is far less than by our present method of treatment; then I think this is going to be a very valuable contribution. When this work was started, I was just as skeptical as Dr. Welles about what could be accomplished. We have listened to Dr. Medlar, we have seen the sections, we have seen the results; and I think most of us who have been following it for some time can be classified as converts. It remains to be seen whether subsequent events will justify our optimism.

There is just one other thing I should like to point out—and I think most of us have been struck by it—namely, the conservation of pulmonary function in Dr. Ryan's cases. One of those he presented had a preoperative maximum minute ventilation of 190 liters and, after multiple resections on both sides, this patient still had 172 liters. That is extremely high. He presented several others with similar findings, but not as high as this one.

These findings are in contrast to the loss of function following thoracoplasty, particularly bilateral thoracoplasty when loss is likely to be extreme. Even in resection I am sure we have all sacrificed too much functioning lung tissue. Several years ago I performed a pneumonectomy for tuberculosis, and in retrospect I am sure that was too much, and even a lobectomy would have been too much. It is entirely possible that a segmental, or even a subsegmental or wedge, would have been sufficient; yet I have seen this same mistake made elsewhere. I am sure many of us in the past have erred by sacrificing far more lung than necessary, both by collapse and by excision.

In summary, this work holds great promise, and if the late results are comparable to the early results, it may revolutionize our thinking on the treatment of tuberculosis.

DR. EDWARD J. O'BRIEN, Detroit.—I am very much impressed with this paper and, as a matter of fact, I am somewhat of a convert myself since listening to it. What impressed me was the fact that antibiotics and collapse have been used for a considerable period before resection. The tendency, in many clinics, is to resect every lesion as soon as it is seen, although a lot of them, we know, do get well and stay well with antibiotics and collapse; but if, after a course of treatment, lesions are standing still or getting worse, I think they should be resected. So-called repeaters should also be resected. One of my greatest hopes is that I may live ten years so that I may see some of this chaos cleared up. Right now it is difficult to get a patient out of a hospital or sanatorium without resection of some sort. The medical men have become very bloodthirsty. You have to be quick on your feet to get a patient discharged with his lungs intact. We, and others, have had a large number of patients get well in the past with only collapse measures, and I am sure will again in the future, but there is such a wide divergence of opinion at present that the situation is chaotic. Opinions vary from resecting every lesion as soon as it is seen, to collapsing a whole lung, and adding paraffin and other packs. If a small caseous center is always going to be such a menace, it is difficult for me to understand how an extensive lesion, that has been collapsed by thoracoplasty, etc., can become healed so frequently, and the patient remain well. We shall have to find a middle ground somewhere, and I think we shall. Medlar's contribution is one of the finest I have heard before this Association, and I think we should take it very seriously. However, I do not believe we shall become as radical as many of the medical men are at the present time, and take out every lesion as soon as we see it.

DR. LEO ELOESSER, San Francisco.—May I ask Dr. Ryan whether his patients, at the end of these resections, turned tuberculin-negative?

DR. RYAN (Closing).—I would like to thank all the discussors. In answer to Dr. Eloesser's question, we have not been testing these patients, but we are certainly going to do so, especially those with minimal lesions; we are going to make tuberculin tests on them in the near future.

With regard to the relapse rate in minimal tuberculosis, our experience is much different from that of Dr. Head. Dr. Roger Mitchell recently studied all the patients discharged from Trudeau Sanatorium in the past seventeen years with minimal tuberculosis. More than 30 per cent of those patients have relapsed.

# A STUDY TO DETERMINE THE EFFECT OF PREVENTION OF OVERDISTENTION OF THE REMAINING LUNG TISSUE ON THE ELEVATED RIGHT VENTRICULAR PRESSURES, FOLLOWING THE RESECTION OF LUNG TISSUE IN DOGS

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MANY investigators have worked on the problems of the lung circulation. Tigerstedt[1] gives in his book concerning the physiology of the circulation an extensive report of the many experiments which have been performed since about a century ago. It is generally accepted that the lung contains more blood during inspiration than during expiration, and least when it is overinflated or compressed. The blood flow is the best during inspiration, since the width of the pulmonary capillaries is then larger than during expiration.

Wasson[2] gives a brief review of the more recent literature about this subject, and it is still agreed that the blood flow through the expanded lung is better than through the collapsed one.

Björk and Salén[3] showed in a study that the blood flow through an atelectatic area of the lung is continuously decreasing during the first month, until practically no blood is passing through the nonventilation part of the lungs. At the same time there is no decrease in size of the main branches of the corresponding artery. Therefore, the blood flow is thought to be shut off in the capillary bed, which is gradually closing down.

Removal of lung tissue reduces the size of the pulmonary vascular bed. The right heart, therefore, has to work against a higher resistance. As a result of this there will be a rise in the pressure in the pulmonary artery and the right ventricle. The more reduction of lung tissue, the higher the resistance to the blood flow in the pulmonary vascular bed will be, and the greater the strain on the right heart. All this has been experimentally proved (among others by Carlson and associates[4]).

The resistance in the pulmonary circulation depends on: (1) the number of vessels, and (2) the diameter of the vessels. If marked overdistention of lung tissue would reduce the diameter of the pulmonary vessels, then it would cause an increased resistance in the pulmonary circulation and impose a greater strain on the right heart. Prevention of marked overdistention then should be considered when lung tissue is to be removed.

It was thought that marked overdistention of lung tissue would stretch the capillaries and thus narrow them, so increasing the resistance and the burden on the right ventricle. Prevention of overdistention therefore might

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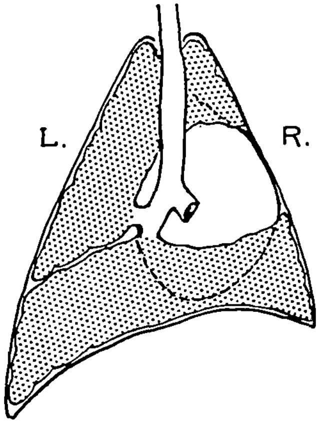
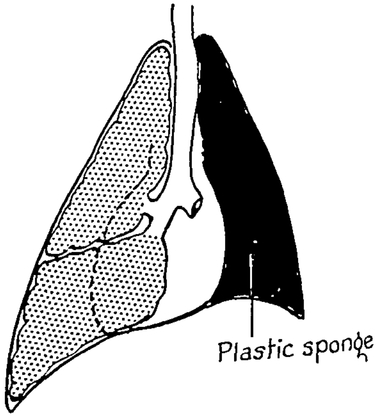
keep the vessels in the more normal shape and might cause less rise in resistance in the pulmonary vascular bed and less strain on the right ventricle.

Following pneumonectomy right ventricular pressures are increased. If overdistention of the remaining lung produces a further increase in right ventricular pressures, one might conclude that there is more resistance and that the width of the pulmonary vascular bed is smaller.

After these preliminary thoughts the following experiment was performed.

Table I. Right Ventricular Pressures in Mm. Hg Two to Three Months After Right Pneumonectomy





| LEFT LUNG OVERDISTENDED | | LEFT LUNG NOT OVERDISTENDED | |
|---|---|---|---|
| DOG NO. | RIGHT VENTRICULAR PRESSURE (MM. HG) | DOG NO. | RIGHT VENTRICULAR PRESSURE (MM. HG) |
| 36 | 43/2 | 821 | 49/5 |
| 379 | 48/4 | 795 | 38/6 |
| 394 | 48/8 | 815 | 39/6 |
| 436 | 48/4 | 602 | 30/1 |
| 437 | 53/11 | 612 | 46/3 |
| 486 | 63/8 | 362 | 31/4 |
| | | 388 | 25/1 |
| | | 534 | 22/2 |
| | | 64 | 42/4 |
| | | 966 | 38/3 |
| Average pressure | 50/6 | Average pressure | 36/4 |

## TECHNIQUE

Ten healthy mongrel dogs of both sexes, with an average weight of 12 kg., were used. A right pneumonectomy was performed with the dissection technique. To prevent overdistention of the remaining lung, a plastic sponge,* as used by Grindlay,[5] was placed in the right thoracic cavity. Following operation the right ventricular pressure was measured at intervals, and the position of the mediastinum was checked by fluoroscopy. The pressures obtained were compared with those from an earlier experiment in which right pneumonectomies were performed on dogs, the remaining lung becoming overdistended.[4] The results may be seen in Table I. It can be seen that the right ventricular pressures in all dogs where overdistention is prevented (except 821 and 612) are lower than in the ones with overdistention, the average systolic right ventricular pressure being lower by 28 per cent (50/6 and 36/4 mm. Hg).

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*This "Ivalon" plastic sponge derived from formalinized polyvinyl alcohol, was obtained from Ivano Inc., Chicago, Ill.

After these results were obtained, it was thought that some of these dogs would be able to tolerate a further reduction of lung tissue, especially since some of the dogs in the group with overdistention survived with only the left upper lobe remaining.

Four months after a right pneumonectomy all ten dogs were subjected to a left lower lobectomy. Intrapleural pressures were adjusted to 0 to −14 cm. water pressure, and no prothesis was used to prevent overdistention during this stage. Right ventricular pressures were studied during this procedure.

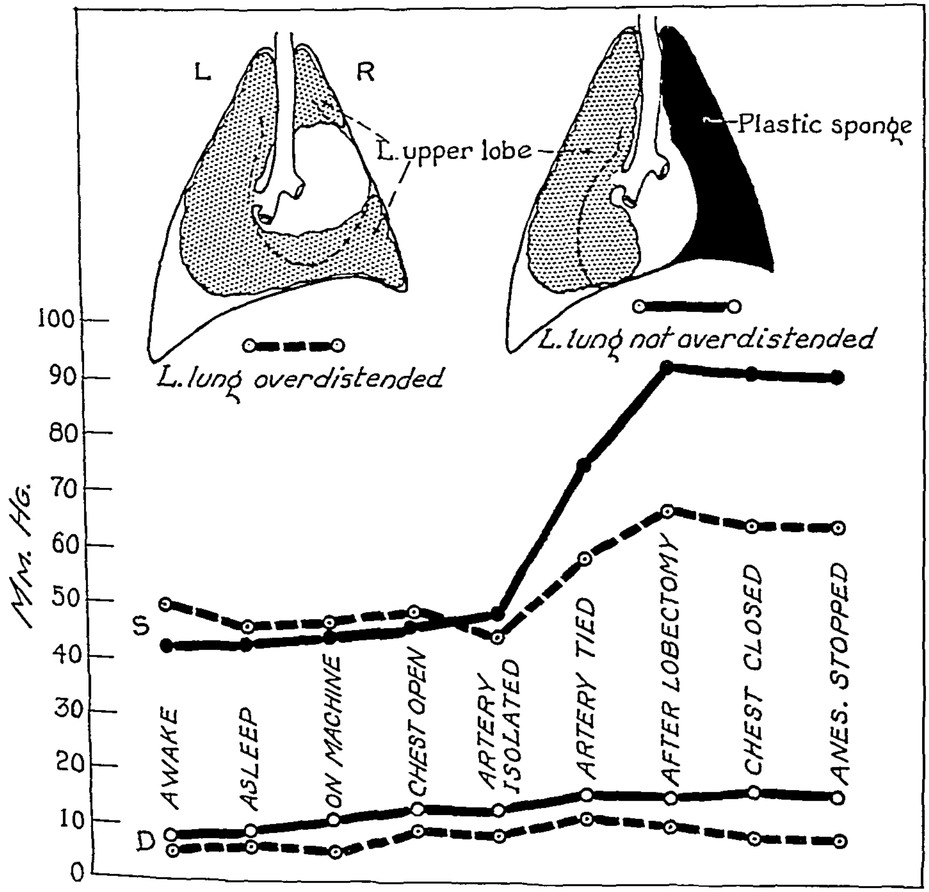


Fig. 1.—Right ventricular pressures during removal of left lower lobe in previously pneumonectomized dogs. There were fifteen dogs in the overdistended group; five survived more than three days. In the not overdistended group there were ten dogs; all died within eighteen hours. Note that in this group the systolic right ventricular pressures are much higher after ligation of the artery as compared to that of the overdistended group.

## RESULTS

At this second stage it was found that at the time of ligation of the pulmonary artery branch to the left lower lobe an immediate increase in the right ventricular pressure occurred that was much higher than in the dogs of Group I where overdistention was present, as can be seen in Fig. 1 and Tables III and IV.

Table II. Removal of All Lung Tissue Except Left Upper Lobe in Stages With Overdistention of Remaining Lung Tissue in Thirteen Dogs. Periods of Survival and Causes of Death

| DOG NO. | SURVIVAL TIME | CAUSE OF DEATH |
|---|---|---|
| 4 | 2 hours | *Cardiorespiratory failure* |
| 36 | Longer than one year | Sacrificed |
| 379 | 2 hours | Cardiorespiratory failure |
| 394 | 16 hours | Cardiorespiratory failure |
| 436 | 1 hour | Embolus, caused by blood transfusion through cardiac catheter |
| 437 | 3 days | Bronchopneumonia |
| 486 | 2 weeks | Sacrificed (was in excellent condition) |
| 593 | 18 hours | Cardiorespiratory failure |
| 256 | 6 hours | Cardiorespiratory failure |
| 26 | 1 hour | Hematoma right heart, caused by cardiac catheter |
| 104 | 2 hours | Cardiorespiratory failure |
| 999 | 5 weeks | Bronchopneumonia |
| 909 | 14 days | Spontaneous pneumothorax |

Table III. Right Ventricular Pressures in Mm. Hg. Left Lower Lobectomy in Dogs Having Previous Right Pneumonectomy. Overdistention of Remaining Left Upper Lobe

| DOG NO. | DOG AWAKE | DOG ASLEEP | ON ANES. MACHINE | CHEST OPEN | ARTERY ISOLATED | ARTERY TIED | AFTER LOBEC. | CHEST CLOSED | ANES. STOPPED | LIVING OR DEAD |
|---|---|---|---|---|---|---|---|---|---|---|
| 4 | – | 40/1 | 43/6 | 44/5 | 45/8 | 52/12 | 58/18 | 54/8 | 55/8 | D |
| 36 | 43/2 | 45/1–2 | 50/–2 | 42/4 | 51/3 | 80/9 | 56/9 | 80/2 | 80/0 | L |
| 379 | 48/4 | 41/5 | 44/6 | 46/12 | 48/12 | 56/15 | 52/6 | 55/6 | 60/9 | D |
| 394 | 48/8 | – | 48/4 | 42/5 | 48/8 | 76/15 | 86/18 | 73/10 | 74/12 | D |
| 436 | 48/4 | 34/17 | 40/6 | 34/12 | 41/12 | 54/16 | 90/11 | 70/16 | 63/8 | D |
| 437 | 53/11 | 58/10 | 52/5 | 38/10 | 36/11 | 42/14 | 72/11 | 63/8 | 64/8 | D |
| 486 | 63/8 | 59/7 | 53/6 | 43/15 | 46/8 | 60/9 | 63/8 | 68/6 | 63/8 | L |
| 593 | – | 42/2 | 41/5 | 45/6 | 42/5 | 53/8 | 55/8 | 52/9 | 53/8 | D |
| Av. | 50/6 | 46/7 | 46/5 | 42/9 | 45/8 | 59/12 | 67/11 | 64/8 | 64/8 | |

Table IV. Right Ventricular Pressures in Mm. Hg. Left Lower Lobectomy in Dogs Having Previous Right Pneumonectomy. Partial Prevention of Overdistention of Remaining Left Upper Lobe

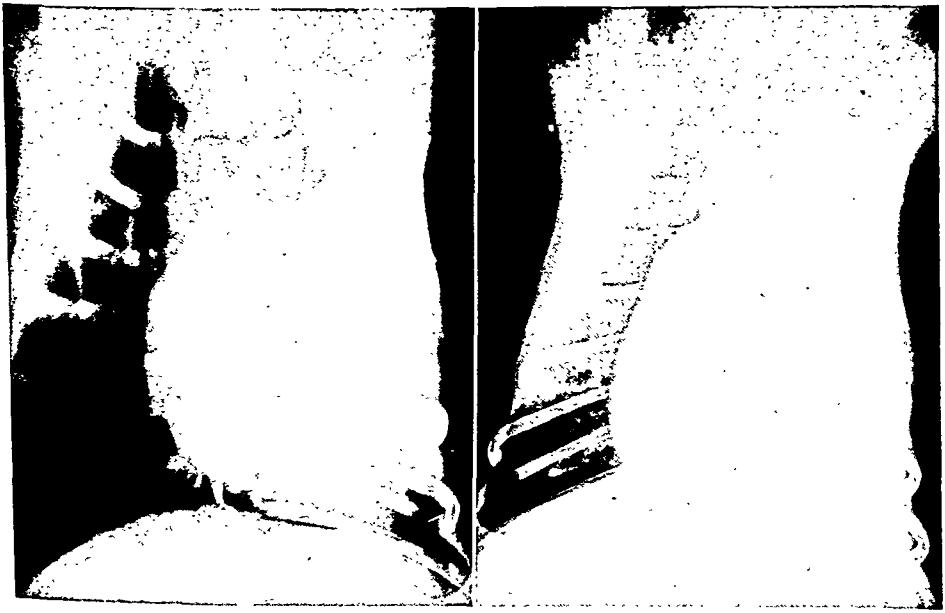
| DOG NO. | DOG AWAKE | DOG ASLEEP | ON ANES. MACHINE | CHEST OPEN | ARTERY ISOLATED | ARTERY TIED | AFTER LOBEC. | CHEST CLOSED | ANES. STOPPED |
|---|---|---|---|---|---|---|---|---|---|
| 821 | 48/4 | 34/17 | 42/6 | 38/8 | 48/12 | 74/16 | 90/11 | 92/16 | 89/10 |
| 795 | 42/5 | 45/13 | 45/9 | 50/14 | 56/10 | 78/16 | 100/15 | 95/15 | 92/13 |
| 815 | 50/7 | 45/5 | 52/6 | 57/9 | 56/9 | 77/11 | 95/12 | 90/13 | 90/14 |
| 966 | 39/3 | 30/2 | 30/2 | 30/2 | 30/2 | 44/6 | 67/9 | 67/9 | 67/9 |
| 602 | 36/7 | 36/7 | 36/5 | 35/6 | 35/6 | 55/9 | 68/6 | 72/4 | 73/4 |
| 612 | 78/27 | 78/27 | 74/44 | 60/45 | 68/42 | 120/50 | 135/55 | 145/55 | 141/51 |
| 362 | 40/15 | 40/15 | 50/15 | 50/15 | 45/15 | 80/15 | 62/10 | 75/15 | 76/15 |
| 388 | 35/0 | 35/0 | 42/10 | 50/20 | 50/20 | 70/25 | 130/25 | 100/25 | 100/25 |
| 534 | 35/3 | 35/3 | 40/5 | 45/10 | 45/10 | 60/15 | 85/10 | 85/15 | 85/15 |
| 64 | 19/–1 | 50/5 | 43/7 | 51/5 | 150/5 | 100/5 | 106/10 | 96/2 | 95/2 |
| Av. | 42/7 | 43/9 | 45/11 | 47/13 | 48/13 | 76/17 | 94/15 | 92/17 | 91/16 |

All dogs survived the operation and were placed in an oxygen box. Immediately following operation they appeared to be in fairly good condition. All of them, however, developed pulmonary edema of which they died within eighteen hours after surgery.

At post-mortem examination it was found that pulmonary edema was present in all, and acute dilatation of the right heart in some dogs. It was also found that the plastic sponge was reduced in size and was covered with fibrous tissue. It was very firmly adherent to the surrounding tissues, especially the pericardium, and was as hard as a stone. No infections were found.

## DISCUSSION AND CONCLUSIONS

It is generally accepted that the resistance in the pulmonary circulation is less when the lung is normally extended as it is during inspiration. This does not necessarily mean that this is true for a markedly overdistended lung, as is the case after a pneumonectomy in a dog. The fact that the right ventricular pressure is lower where overdistention was prevented makes us believe that overdistention above a certain degree causes higher resistance and imposes a greater strain on the right ventricle.



*A.* *B.*

Fig. 2.—Roentgenograms of two dogs following right pneumonectomy: *A*, with subsequent overdistention of the remaining lung. *B*, right pleural cavity filled with plastic sponge to prevent overdistention. Note less shift of mediastinum, with less overdistention of remaining lung.

After this theory one would expect more surviving animals in the group of dogs where overdistention was prevented than in the overdistended group where 85 per cent lung tissue was removed and where there were four of thirteen animals surviving for longer than two weeks (Table II). The expectation was wrong, as all dogs died. The fact that there was no overdistention in this group probably is just the cause of death, the pulmonary vascular bed not having had a chance to overdistend gradually, as probably is the case where overdistention is not prevented. (The right ventricular pressure came down after several weeks in pneumonectomized dogs where overdistention was not prevented.[4])

If, then, after a pneumonectomy the pulmonary vascular bed is again suddenly reduced by more than 50 per cent, the remaining lung tissue (left upper lobe in this experiment) does not have enough vessel capacity to transport all the blood, while in the overdistended left lung the vessels have had more chance to increase gradually in size over a longer period of time.

We believe that 50 per cent reduction of lung tissue and prevention of overdistention of the remaining 50 per cent will impose less strain on the right heart, but when further reduction of lung tissue is anticipated, overdistention might be favorable to give the remaining lung tissue and pulmonary vascular bed a chance to increase in size over a longer period of time, so that at the time of the next operation the remaining very small amount of lung tissue is able to handle the total blood flow.

Collapse of a lung by pneumothorax, prior to surgery, to be resected should enable the patient to become adjusted to the use of one lung. The beneficial effects of a pneumothorax given before a pneumonectomy in patients might partially be explained by the results of this study.

The plastic material used in this experiment is not a satisfactory type of material to use as a prothesis in the chest, since it causes too many adhesions and is too adherent to the pericardium, thus perhaps interfering with the heart action. On the basis of these experiments, it would seem to be definitely undesirable for use in patients.

Since dogs have an unstable mediastinum, a pneumonectomy will cause marked overdistention of the remaining lung. In patients, however, a more or less stable mediastinum usually prevents too much overdistention.

Bilateral lobectomy, however, might very well cause more rise in right ventricular pressure because of overdistention of the remaining lobes. Prevention of overdistention here might be of some help in hazardous cases. It is, however, well known that most young patients tolerate a bilateral lobectomy very well.

Rasmussen and associates stated in 1941 after an experimental study: "from the above findings it would appear that temporary paralysis of the diaphragm is a justifiable procedure in the human being. In bilateral operations it seems advisable that sufficient time elapse between stages for re-establishment of the diaphragmatic function and improvement of the patient's general health." To this might be added: "and adjustment of the pulmonary vascular bed."

## SUMMARY

1. Approximately 85 per cent of lung tissue was removed in stages in ten dogs, while overdistention of the remaining left upper lobe was partially prevented by inserting a plastic sponge in the thoracic cavity. The right ventricular pressures were studied.

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Since dogs have an unstable mediastinum, a pneumonectomy will cause marked overdistention of the remaining lung. In patients, however, a more or less stable mediastinum usually prevents too much overdistention.

Bilateral lobectomy, however, might very well cause more rise in right ventricular pressure because of overdistention of the remaining lobes. Prevention of overdistention here might be of some help in hazardous cases. It is, however, well known that most young patients tolerate a bilateral lobectomy very well.

Rasmussen and associates stated in 1941 after an experimental study: "from the above findings it would appear that temporary paralysis of the diaphragm is a justifiable procedure in the human being. In bilateral operations it seems advisable that sufficient time elapse between stages for re-establishment of the diaphragmatic function and improvement of the patient's general health." To this might be added: "and adjustment of the pulmonary vascular bed."

## SUMMARY

1. Approximately 85 per cent of lung tissue was removed in stages in ten dogs, while overdistention of the remaining left upper lobe was partially prevented by inserting a plastic sponge in the thoracic cavity. The right ventricular pressures were studied.

2. Prevention of overdistention of lung tissue in pneumonectomized dogs reduces the amount of elevation in the right ventricular pressure following that operation.

3. Removal of all lung tissue except the left upper lobe caused death of all dogs, while in an experiment where overdistention was not prevented some dogs survived for weeks, with only the left upper lobe remaining.

4. The plastic sponge (Ivalon) used in this experiment is not a satisfactory material to use as a prothesis in the chest, and should not be used in patients, since it becomes adherent to the pericardium and might interfere with heart action.

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# TECHNIQUES OF ENDOCARDIAL ANASTOMOSIS FOR CIRCUMVENTING THE PULMONIC VALVE

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THE background and rationale for this approach to the surgical treatment of pulmonic stenosis and atresia have been reviewed in a previous publication by one of the authors. It has been pointed out that the nature and varying combination of defects in pulmonic stenosis and atresia make a direct attack on the infundibular type of stenosis less feasible than a shunting procedure.[1, 2] The latter technique provides a good outflow tract for the right ventricle with the least residual derangement in cardiodynamics. In that previous investigation, one end of an homologous vein graft was anastomosed to the distal segment of the ligated left pulmonary artery after which the other end of the graft was everted over a polyethylene tube and inserted into the right ventricle (Fig. 1). These new left pulmonary arteries remained patent for two to three weeks, and were then thrombosed. While patent, the vein valves functioned to prevent regurgitation, and the shunt delivered blood in much the same manner as the original left pulmonary artery. It has been found that homologous vein grafts substituted for an excised segment of left pulmonary artery remain permanently patent (Figs. 2 and 3). Thus, the tube anastomosis at the cardiac end of the shunt is the vulnerable portion from the standpoint of thrombosis.

Since the myocardium partially closes openings in the ventricular wall during systole, and because suture anastomoses to endocardium become obliterated, a rigid tube of some kind is necessary to hold the ventricle open and permit blood flow through the shunt.[1, 3] This rigid tube can be designed with a flare approximately perpendicular to its wall to provide immediate hemostasis when inserted into the ventricle, thus making it unnecessary to interrupt the circulation in completing the shunt. Lining the rigid tube with a vein facilitates intima to endocardial healing and presents smooth endothelium to the bloodstream. Since the initial venous-lined polyethylene tubes failed to remain permanently patent, various types of rigid tubes were substituted. It was hoped that a less reactive material might better the anastomosis in much the same manner that Blakemore, by using the relatively nonreactive vitallium, improved on Nitze's ivory rings and the magnesium

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tubes of Payr.[4-6] The types of tubes used were polyethylene, lucite, teflon, and Kel F plastics, and homologous tracheal grafts prepared in three different ways. Only the series of venous-lined tracheal and polyethylene tubes will be described in this report. The other plastics used with and without venous lining will be alluded to briefly but described in detail in another publication.

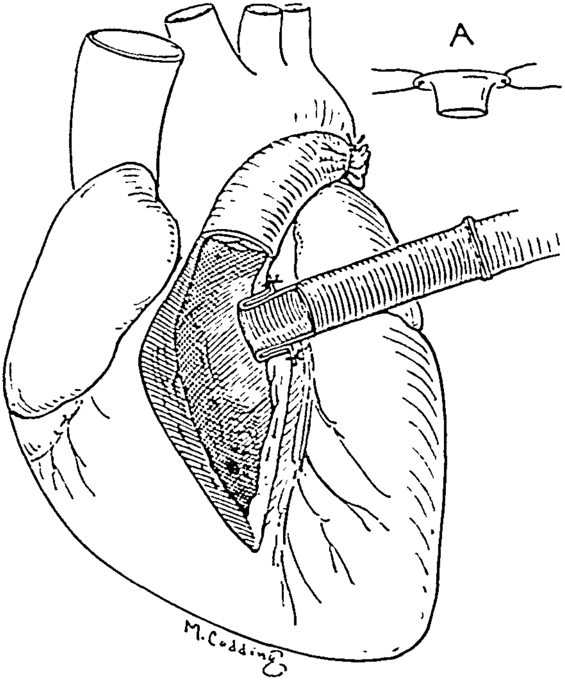

Fig. 1.—Cross section of venous-lined tube in the right ventricle. Left pulmonary artery is ligated just beyond the bifurcation of main pulmonary where right pulmonary artery courses under aortic arch. Distal left pulmonary artery is sutured to the homologous vein graft as depicted. Insert A shows the plastic tube without vein graft.

## MATERIALS AND METHODS

*Preparation of the Vein Grafts.*—The grafts were abdominal cavae excised from the liver to below the renal veins. The donor dog was less than two hours post mortem, and about 3 kg. heavier than the recipient, of similar nutritional status, in order to match the graft and the left pulmonary artery at the suture anastomosis and to avoid stretching or "funneling" at the tube anastomosis. The veins were taken with aseptic precautions and stored for periods up to seventeen days in solution I at 0° to 10° C.*

*Preparation of the Tracheal Tubes.*—The tracheas of recently sacrificed dogs were excised 1 cm. above the carina and a few millimeters below it on the right- and left-main stem bronchi. The medial walls of the bronchi were excised, leaving a semiflexible flare at one end of the tube of trachea. The donor was selected about 6 kg. smaller than the recipient dog to match the trachea with the donor vein (chosen as described previously) and to provide an ample stoma in the heart wall. The tracheal grafts were prepared in three ways. The first group was immersed in a solution of 90 c.c. isotonic saline and 10 c.c. of 40 per cent neutral formalin. After three to twelve hours they were stored in the same solution diluted ten times. Twenty-four hours before operation they were washed repeatedly in sterile distilled water to remove all but the protein-bound formaldehyde. The second group of tracheas was mechanically cleaned with phisoderm and G-11 and after thorough rinsing immersed in a solution of 80 c.c. of balanced salt solution buffered with 1.4 per cent bicarbonate and 20 c.c. of 1:1000 zephiran and stored in a refrigerator at 0° to 10° C. The third group was mechanically cleaned with the phisoderm and G-11 mixture and stored at 0° to 10° C. in Solution I, the solution used to preserve the vein grafts.

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*Solution I consists of 85 per cent balanced salt solution, with bicarbonate buffer and phenol red indicator, homologous serum 10 per cent, and antibiotics 5 per cent. The formula for balanced salt solution, which is similar to Tyrode's solution, can be found in the publication of Pierce and associates.[7] The antibiotics in the above solution were penicillin 400 units per cubic centimeter, streptomycin 2 mg. per cubic centimeter and sulfadiazine 0.12 mg. per cubic centimeter. Sulfadiazine was added to insure the sterility of the tracheal grafts stored in Solution I. For the vein grafts taken with aseptic technique, the antibiotics were probably superfluous.

Portions of the tracheal and venous grafts were taken for bacteriologic study, while other specimens were incubated in tissue culture medium to determine preoperative viability. Each bacteriological specimen was thoroughly washed in sterile distilled water to remove antiseptics and antibiotics.

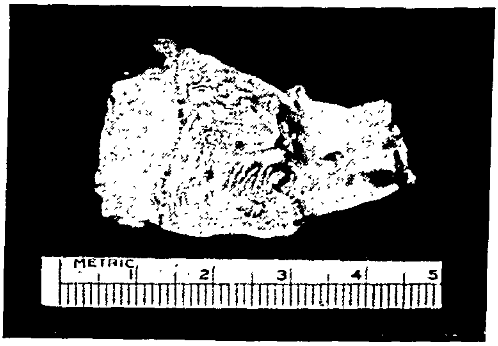

Fig. 2.—A segment of homologous vena cava substituted for an excised segment of left pulmonary artery. Proximal suture line is at one centimeter on the ruler; distal anastomosis at three centimeters. Note smooth endothelial lining of the graft and suture lines at twenty-seven days postoperatively.

*Preparation of the Polyethylene Tubes.*—Stock tubing of 11 mm. inside diameter and 12.5 outside diameter was cut into lengths of 14 mm. and one end flared with a hot die. A few millimeters from the opposite end a groove was made around the circumference. The effective length of the tube from this end to the flare was about 10 mm. or 12 to 14 mm. including the flare. The tubes were sterilized in 1:1000 aqueous zephiran for eighteen hours prior to operation.

*Operative Technique.*—The animals were premedicated with 0.6 mg. of atropine sulfate and anesthesia induced with sodium pentothal intravenously. Two polyethylene tubes were inserted through a size 14 needle into foreleg veins and used for subsequent injections of blood, dextrose, or cardiac drugs.[8] A rubber tube which fitted a little loosely was inserted into the trachea and connected to an Erlanger pump. The latter supplied compressed air and ether with valves to modify rate and depth of respiration, the relative lengths of inspiration and expiration, and the concentration of ether in the mixture.

The dog was placed midway between the supine and right lateral positions. The fourth left intercostal space was opened from the sternum to the tip of the scapula, and the fifth rib divided at the sternal junction and under the tip of the scapula. The left pulmonary artery, freed from the hilus of the lung to the bifurcation, was clamped near the hilus and ligated close to the bifurcation. The artery was then divided close to the ligature and cuff reinforced with a suture ligature. The distal segment was irrigated, stripped of adventitia, and the end cut on the bias when the vein graft appeared slightly large. The graft which had been fixed to the tracheal or plastic tube was anastomosed with a continuous everting mattress suture of

00000 arterial silk or chromic catgut to the distal end of the divided left pulmonary artery. Retrograde bleeding and saline irrigations were used to test the anastomosis and vein graft for leaks (Fig. 1).

The pericardium was then opened along the interventricular septum with a short perpendicular extension to the tip of the right auricle. The right edge of the pericardium was marsupialized with several pericardial sutures to rotate the right ventricle to the left of the partially overlying sternum. Four epicardial sutures of 00 cotton were then spaced on the sides of a rectangle running from the pulmonary conus 2 cm. down the ventricular wall and about 1 cm. from the septum. Each of the four sutures was spaced equally through the flare of the tube or trachea in the appropriate position to provide hemostasis and prevent twisting of the vein graft. If the flare or tube was larger than usual a fifth epicardial suture was placed parallel to and just below the pulmonic valve perpendicular to the planes of the other stitches. This suture was removed if the other four provided complete hemostasis.

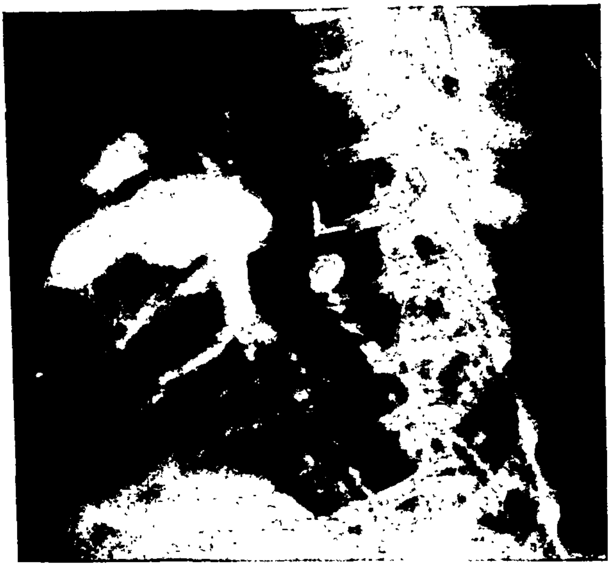

Fig. 3.—Another control graft interposed in left pulmonary artery. Graft was purposely made redundant in length but despite angulation was fully patent at autopsy fourteen months postoperatively. *Angiocardiogram* is 9.5 *months postoperatively*. Arrow points to proximal anastomosis partially obscured by tracheal shadow.

In the first half of the series a Bard-Parker blade was used to make the opening into the ventricle. The only two deaths during operation in the series were due to excessively large incisions in the heart. New blades of various widths were therefore designed with a cutting edge on the end of the blade almost perpendicular to the handle and pointed with a central angle of about 115 degrees.* By inserting the blade of proper width perpendicular to the heart wall, an incision could be made 2 to 3 mm. larger than the tube diameter which would permit the introduction of the tube into the ventricle without resistance and with complete hemostasis following insertion. With proper performance of the heart anastomosis, blood loss, and trauma were so slight that no grossly perceptible change in contractility or blood pressure occurred. If the blood loss was more than 50 c.c. it was quantitatively replaced through the polythene tubes as rapidly as the heart would accept it without dilatation or diminished contractility. If contractility decreased seriously, 10 c.c. of 50 per cent glucose was given intravenously followed by 0.5 c.c. of a solution of 1 c.c. of 1:1000 adrenalin in 10 c.c. of 1 per cent procaine. If this dose given intravenously did not improve contractility, another 0.5 c.c. of the mixture was given into the right auricle. If the adrenalin procaine solution had to be given again later, another injection of 50 per cent glucose was made and maximal aeration of the lungs obtained to prevent ventricular fibrillation. If ventricular

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*The authors are indebted to Rudolph Beaver of The Beaver Mfg. Co., Waltham, Mass., for designing and supplying the knife blades for this work.

TABLE I. SERIES OF OPERATIONS IN SUMMARY FORM

| DOG NO. | TUBE IN HEART ANASTOMOSIS | LENGTH OF SURVIVAL | CAUSE OF DEATH | CONDITION OF DOG | CONDITION OF SHUNT | | |
|---|---|---|---|---|---|---|---|
| | | | | | AT OPERATION | POSTOPERATIVE ROENTGENOGRAMS | AT AUTOPSY |
| 1 | Formalin Trachea | 7.3 months | Sacrificed | Excellent | 2+ | – | 0 |
| 2 | Formalin Trachea | 4.5 months | Sacrificed | Excellent | 2+ | – | 0 |
| 3 | Formalin Trachea | 4.5 months | Sacrificed | Excellent | 3+ | 68 days—0 | 0 |
| 4 | Zephiran Trachea | 6.9 months | Sacrificed | Excellent | 4+ | 36 days—0 | 0 |
| 5 | Zephiran Trachea | Operative death | Hemorrhage | – | – | – | – |
| 6 | Zephiran Trachea | Operative death | Hemorrhage | – | – | – | – |
| 7 | Zephiran Trachea | 8 days | Pneumonia | – | 2+ | – | 0 |
| 8 | Zephiran Trachea | 3.5 months | Sacrificed | Excellent | 3+ | 14 days—0 | 0 |
| 9 | Zephiran Trachea | 6 days | Pneumonia | – | 3+ | – | 4+ |
| 10 | Solution I Trachea | 3.5 months | Sacrificed | Excellent | 4+ | 22 days—4+<br>43 days—0 | 0 |
| 11 | Solution I Trachea | 5.6 months | Sacrificed | Excellent | 4+ | 29 days—2+<br>37 days—0 | 0 |
| 12 | Solution I Trachea | 2.6 months | Sacrificed | Excellent | 3+ | 28 days—4+<br>34 days—4+<br>43 days—3+<br>59 days—0 | 0 |
| 13 | Solution I Trachea | 2.6 months | Sacrificed | Excellent | 3+ | 28 days—0 | 0 |
| 14 | Polyethylene | 27 days | Sacrificed | Distemper | 4+ | – | 2+ |
| 15 | Polyethylene | 1.3 months | Sacrificed | Excellent | 4+ | 21 days—0 | 0 |

Under "condition of shunt" 4+ denotes maximal filling, 3+ indicates good filling of a fully patent shunt, 2+ represents definite obstruction to filling, 1+ denotes marked obstruction and 0, complete occlusion of shunt.

TABLE II. RESULTS OF TREADMILL STUDIES ON SECOND TRIAL OF DOGS STOPPED WELL SHORT OF MAXIMUM EFFORT

| DOG NO. | POSTOPERATIVE INTERVAL | TYPE OF CARDIAC TUBE | DEGREES OF INCLINATION OF TREADMILL | SPEED (M.P.H.) OF TREADMILL | TIME ON TREADMILL IN MINUTES | CONDITION IMMEDIATELY AFTER RUN |
|---|---|---|---|---|---|---|
| 1 | 3.8 months | Formalin Trachea | 10 | 4.2 | 17 | Pulse 128. Frisky |
| 8 | 2.5 months | Zephiran Trachea | 10 | 4.2 | 32 | Pulse 136. Strong |
| 11 | 2.0 months | Solution I Trachea | 10 | 4.3 | 19 | Running easily |
| 15 | 24 days | Polyethylene | 10 | 4.3 | 20 | Running well but pulse 160 and panting rapidly |

Although dog number 15 was too early postoperatively to have developed full strength or abundant collateral circulation through the bronchial arteries, he ran well without signs of exhaustion.

fibrillation supervened, manual massage was instituted until the heart could be shocked into asystole with a 1 to 1.5 ampere current and the above measures instituted to restore contractility.[9]

The chest was closed in layers with careful attention to the expansion of the left lung and the removal of intrapleural air through catheter suction during closure. If the breath sounds were diminished following closure, the pleural space was aspirated with a needle, and if they were associated with râles and rhonchi the trachea was aspirated and, if necessary, bronchoscopy performed. Crysticillin was given for a few days postoperatively.

*Postoperative Observations.*—Angiocardiography was performed at intervals postoperatively to determine patency, and function of the graft and treadmill studies were used to evaluate the general cardiopulmonary function. The animals were sacrificed under nembutal and histologic study made of the significant gross changes at autopsy.

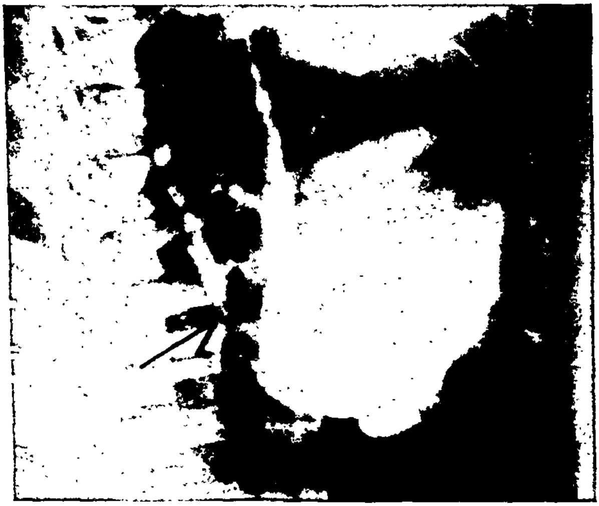

Fig. 4.—Dog No. 12 on the thirty-fourth postoperative day. A similar film was obtained on the forty-third day. Air in the trachea partially obscures the graft (arrow), but patency of the shunt and filling of the left pulmonary arterial tree are evident.

## RESULTS

*Toleration of the Operative Procedure.*—Table I summarizes the results of fifteen operations. The two deaths during operation occurred before the use of special heart knives to control the size of the heart incision. The two other deaths were about one week postoperatively from atelectasis and pneumonia. Both followed anesthesias characterized by profuse mucous secretions and diminished respiratory excursions during and immediately after operation. Preoperative atropine and, if necessary, postoperative suction or bronchoscopy have eliminated this complication in forty subsequent cardiac operations. The operative mortality in this series including the anesthetic complications would be 27 per cent, while the mortality from the cardiac procedure per se would be 13 per cent.

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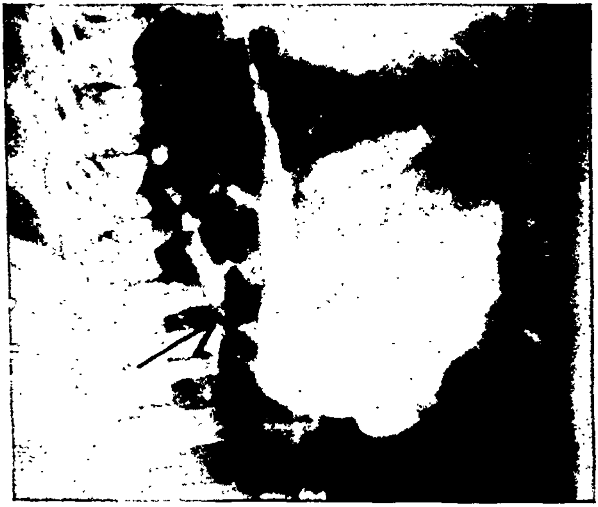

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*Function of the Vein Grafts.*—Table I shows the condition of the shunt at operation on various postoperative days and at autopsy. All the grafts with the exception of dogs 1, 2, and 7 filled well at operation, giving a pink lung and normal pulsations in the distal left pulmonary artery. The filling of the grafts in dogs 1, 2, and 7 was diminished because of tubes too short, too narrow, or only partially through the ventricular wall. All the grafts, however, were occluded at autopsy with the exception of the two animals that died on the sixth and twenty-seventh postoperative days. Roentgenograms taken at various postoperative intervals showed one shunt open at forty-three days postoperatively (Fig. 4). Although some grafts were open at three weeks, most were closed before the end of the fourth postoperative week.

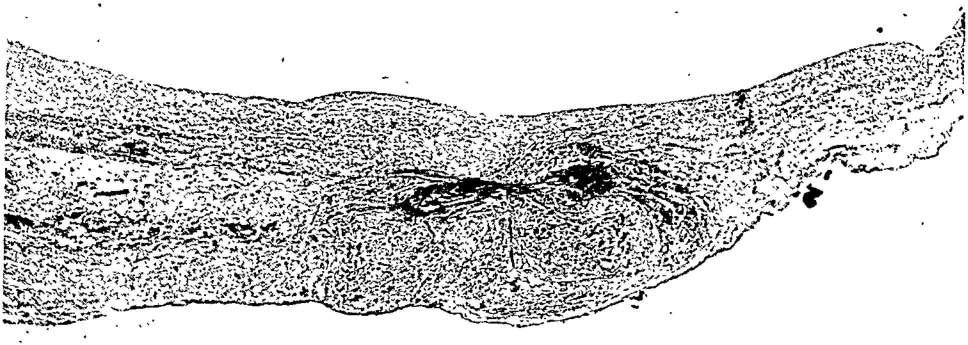
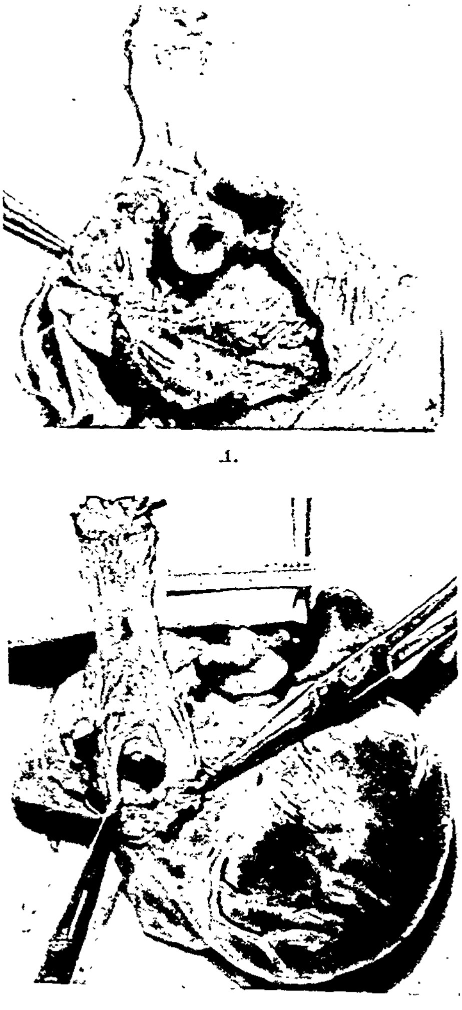


Fig. 5.—Photomicrograph of the suture anastomosis of dog No. 14 on the twenty-seventh postoperative day. Note endothelioid lining of the graft and granulomatous response to arterial silk, especially on the narrow or graft side of the anastomosis.

*Autopsy Findings.*—In all cases, except the two that died of pneumonia, the lungs were pink, crepitant, and expanded. The two exceptions had partially collapsed and consolidated left lungs which sank in water. Microscopically they showed pneumonia and some agonal pulmonary edema. The left lungs were almost always adherent to the intercostal incision, the pericardium, and the graft. There was no detectable difference between the reaction to various types of preserved trachea and to the pure polyethylene tubes.[11, 12]

The vein grafts from dogs more than three months postoperative were shrunken, and the walls apposed. The graft, patent at autopsy six days postoperatively in dog No. 9, showed the early host inflammatory reaction in the adventitia. The donor endothelium had disappeared and the reconstruction of the intima had hardly begun. This left bare areas of subendothelial connective tissue in contact with the blood stream.[13] This surface, on gross examination, looked reddish and had tiny clumps of fibrin in various areas of the graft. The vein graft in dog No. 4, patent at autopsy on the twenty-seventh postoperative day, showed no fibrin deposition macroscopically, and a firm intimal union at the suture anastomosis on microscopic study. The intima became thinner towards the middle of the graft, but was lined for the most part with smooth endothelioid cells. The round-cell inflammation of the adventitia had almost completely subsided without as yet much fibrous tissue reconstruction. The lack of fibroplasia in the adventitia and media plus the disappearance of donor tissue in these layers left the wall thin at this stage of healing (Fig. 5). The endocardial end of this shunt was typical of most of the venous-lined plastic tubes, as reported previously.[1] Just inside the intraventricular portion of the tube a fibrous constriction of the vein graft reduced blood flow to a minimum and would have produced thrombosis distally in a few days (Figs. 6 and 7). Histologic sections of this area showed serosanguineous exudate between the



A.

B.

Fig. 6.—*A*, Endocardial, and *B*, epicardial views of the shunt in dog No. 14, twenty-seven days postoperatively. The typical fibrous constriction of the graft just inside the tube has narrowed the lumen to about 3 mm. in that area and would have precipitated thrombosis in a few days. Note well healed suture anastomosis.

adventitia of the lining vein and the inner surface of the plastic tube. In addition to the marked proliferation of connective tissue, there were small areas of necrosis and hemorrhage scattered throughout this portion of the graft wall suggesting continual trauma.

The rate at which the host destroyed the tracheal grafts varied widely and for the most part pari passu with the degree of reaction around the silk sutures. Although the grafts prepared in formalin had slightly thinner and more rigid walls, destruction of the fibromuscular tissue between the tracheal rings deformed all the grafts during the first two months postoperatively, causing gradual collapse and, in a few, extrusion from the ventricle. This extrusion from the ventricle did not occur with the plastic tubes—patent or thrombosed. The destruction and removal of the cartilaginous rings was notably slow and incomplete at six months or even longer. The cartilage cells in the lacunae were shrunken or absent from the outset. The lacunae became closely packed and confluent as the matrix degenerated and disappeared. This left large holes containing the basophilic residue of cartilage undergoing phagocytosis by lymphocytes and macrophages. In other areas, round-cell invasion and isolation of small fragments of cartilage was followed by organization of the acellular matrix by fibrous tissue.

*Bacteriologic and Tissue Culture Studies.*—The tracheal tubes prepared with formalin and zephiran were, of course, sterile and nonviable. Tracheal tubes mechanically cleaned with phisoderm and G-11 soap and immersed for two or more days in Solution I were likewise free of bacterial growth. Bacteriologic cultures of the latter group of tracheal specimens were positive in the few specimens studied, if either the phisoderm and G-11 washing or the immersion in Solution I was omitted. Veins taken aseptically and stored in Solution I were found to be sterile.

Vigorous fibroblast proliferation could be seen in tissue cultures of one- and two-week-old veins. Veins three weeks or more in Solution I failed to show growth in tissue culture. No tracheas prepared in the three ways previously described grew in tissue culture. One specimen of trachea washed in water and kept in Solution I for ten days showed moderate growth.

## DISCUSSION

In the previous report it was noted that the dogs that emerged from anesthesia invariably tolerated the new shunt well—patent or occluded. The operative mortality in those initial experiments was about 50 per cent, although the brevity of the series and the continual change in operative technique made this figure an approximation. Although the technique is still changing, the operative mortality in this series is a definite improvement over that reported earlier. The anesthesias in the initial series were intravenous nembutal supplemented by a mechanical respirator which delivered 100 per cent oxygen but did not eliminate the positive pressure during the expiratory phase and permit adequate collapse of the lungs. In several instances, the resulting hypercapnia and decreased ventricular filling were sufficient to cause death before the anastomoses were attempted. In other operations it caused excessive mediastinal and diaphragmatic motion, thus prolonging the operation as well as lowering the myocardial reserve. With intermittent positive pressure air or oxygen fully expanding and collapsing the lung at a rapid rate, the mediastinum and diaphragm can be kept almost motionless on a light ether anesthesia. This supports myocardial function and facilitates the anastomoses greatly.

The insertion of the intracardiac tube can be done without significant blood loss or trauma if the opening in the heart is the proper size, i.e., 2 to 3 mm. longer than the outside diameters of the tubes. If the knife described previously is sharpened regularly and inserted carefully perpendicular to, and completely through the heart wall, the length of the incision in the endocardium should be about the same as the width of the knife blade. With four or five epicardial sutures spaced properly with both ends through the tube flare, hemostasis is assured even when the heart incision is slightly large. The use of No. 00 cotton assures tight ligation of these epicardial sutures without breakage.

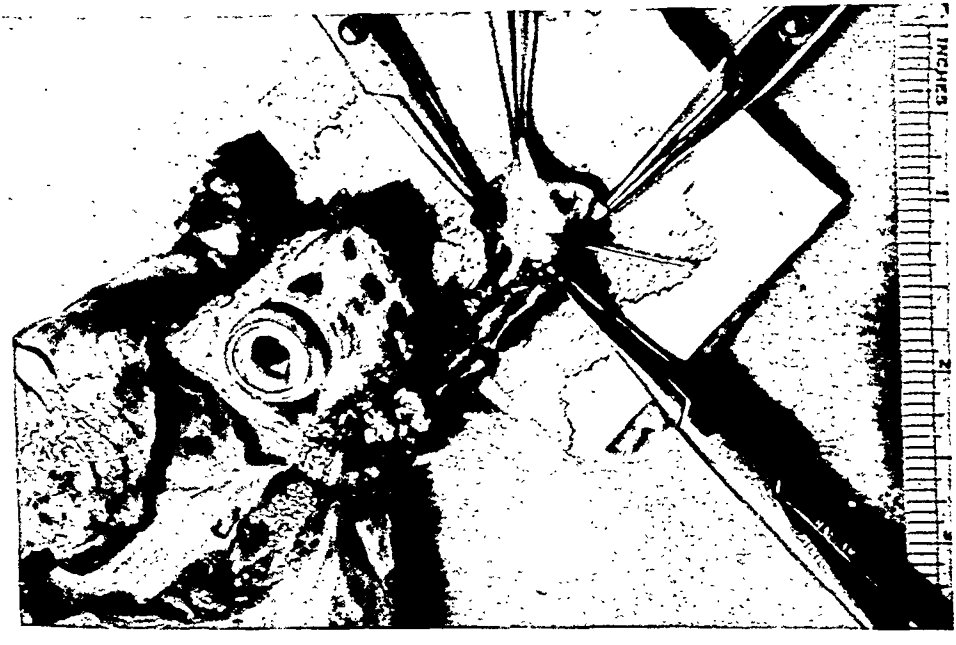


Fig. 7.—The shunt in dog No. 15, 1.3 months postoperatively. The thrombus extends from the constricted area of the graft distally to within one centimeter of the barely visible catgut suture anastomosis. The beginning shrinkage of the graft is typical but the clot usually propagates to and involves the silk suture anastomosis.

Except for the two animals that died of postoperative atelectasis and pneumonia and another animal with distemper, all the animals surviving the operative procedure were healthy and well. The treadmill studies in these animals corroborate the previously reported electrocardiographic, roentgenographic, and other evidence of normal cardiac function in the presence of the intracardiac tube and graft. Even without training, conditioning, and maximal effort the animals did an amount of work incompatible with cardiac decompensation or any marked lowering of myocardial reserve.

The failure of these shunts to remain permanently patent is due to two principal factors: (1) the lack of sufficient pressure gradient on ligation of the left pulmonary artery, and (2) the intrinsic weakness of the venous-lined tube used in the heart anastomosis. It is well established that after ligation of one pulmonary artery, the blood flow is shunted to the opposite lung without significant or prolonged elevation in main pulmonary artery pressure.[14] The development of collateral circulation through the bronchial arteries to the distal left pulmonary artery per se elevates the left pulmonary artery pressure to half of the normal value, further dissipating the pressure gradient and the volume flow through the shunt.[1, 15, 16, 17] It has been shown that the Blakemore type of anastomosis is inferior to suture anastomosis in preventing thrombosis, especially in anastomoses with low pressure gradients.[18-21] When the venous-lined tube is placed in the ventricle, the brunt of the stretching force of each systolic ejection is transmitted to the fixed portion of vein graft near the point of eversion over the end of the tube. This area of tension thickens and progressively restricts flow until thrombosis occurs just beyond the constriction and propagates distally to the suture anastomosis. Another factor favoring constriction of the tube in this area is the collection of serosanguineous exudate in the space between the adventitia of the lining vein and the inner surface of the rigid tube. In the venous-lined tracheal tubes, the gradual distortion and destruction of the tracheal graft by host reaction is enough per se to occlude the heart anastomosis.

It is possible to obviate these deleterious factors by attaching the vein graft to the outer end of the tube. By using various smooth and water repellent plastic surfaces for the intracardiac portion of the shunt, it has been possible to ligate the main pulmonary artery and permanently divert the entire cardiac output through the shunt without clotting or untoward changes in the cardiac function. This work is being reported in another publication.

## SUMMARY

A series of fifteen attempts at constructing a new left pulmonary artery with venous-lined tracheal and polyethylene tubes resulted in eleven survivals which remained healthy and well with the exception of one animal with distemper. There were two operative deaths from hemorrhage and two postoperative deaths from atelectasis and pneumonia. These deaths are considered preventable in the light of present knowledge and refinements of operative technique. Four animals ran on the treadmill at a 10-degree incline for distances within the range for normal animals.

Angiocardiography demonstrated good filling of some shunts as long as forty-three days postoperatively, but the majority of shunts were occluded by the third or fourth week. The factors causing these thromboses have been analyzed, and other types of endocardial anastomoses devised which obviate these factors.

## CONCLUSIONS

1. With attention to principles mentioned herein, a new left pulmonary artery may be constructed in the normal dog with a vein graft and a rigid intracardiac tube with a low operative mortality.

2. The animals surviving the operation will remain healthy—with or without patency of the shunt.

3. With venous-lined tubes of homologous trachea or polyethylene, the shunts eventually became occluded. This usually occurs during the third or fourth week postoperatively.

4. The factors causing this thrombosis have been analyzed and described.

The authors wish to express appreciation to Miss Elnore Hitchcock for assisting in the operative procedures. They are also grateful to Dr. John Hanks for his advice and help in the preservation of the various grafts and in testing their viability in tissue culture. Mr. Rudolph Beaver has kindly supplied the heart knives.

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# FIBROSARCOMA OF THE DIAPHRAGM

## Report of a Case

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PRIMARY tumors of the diaphragm do not occur frequently. A survey of the literature[1-35] disclosed thirty-eight fully substantiated cases of diaphragmatic tumors, including the present case, of which sixteen were classified as benign, while twenty-two were malignant (Table I). The benign tumors, fibromyomas and lipomas, are the most common, while fibrosarcoma is the most often-reported malignant tumor.

Of the tumors described as benign, those of Kinsella[26] and those of Bonamy,[25] closely resemble the benign phase of the case herein described. Kinsella[26] reported a 2-pound fibromyoma from the anterior portion of right hemidiaphragm, occurring one-half above and one-half below the diaphragm, and resembling fibromyoma of the uterus grossly. Bonamy[25] reported 1,200 Gm. of fibromyomas arising from the right side of the diaphragm, with one large tumor and four smaller ones. Recovery followed the surgical treatment in both of these cases. Gale and Edwards[12] suggest that the tumor described by them as "primary endothelial sarcoma" might be benign, and reported that histologic opinions obtained from several competent pathologists varied widely. This is not an uncommon experience, and illustrates that it is admittedly difficult in special cases to draw a sharp line between malignant and benign tumors of the diaphragm.

The patient's history and clinical course aids in making a diagnosis of a mass in the region of the diaphragm. Tumors of the diaphragm caused chest pain in ten of the cases, abdominal pain in one, and related pain in another. Fullness in the lower chest precedes or accompanies dyspnea, especially upon exertion. Edema occurs if there is embarrassment of the heart in advanced cases.

### CASE REPORT

This patient, a white man, aged 60 years, occupation farmer, experienced three attacks of illness in the last ten years, all somewhat similar, but differing in several details. Upon admission, the chief complaint was pain and fullness of the left chest, shortness of breath, and edema of feet and ankles.

The first attack occurred in 1938 when he was suddenly struck by momentary stabbing pains recurring every few minutes, beginning in the abdomen rather deeply, but anteriorly, in the left anterior axillary line approximately one-half distance between the costal margin and iliac crest, and migrating rapidly up the left side of the chest, over the left shoulder, and then down the left posterior chest to reach the point of origin. This attack covered a one-week period. Later pains became worse with deep breathing, and severe pain tended to center in the "heart region." He doubted his ability to survive the next pain. Shortness of breath rapidly developed, and was especially aggravated by exertion. At the onset he took

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From Boehne Tuberculosis Hospital.
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strong physic with whiskey for a "stimulant," and the resultant caused fullness in the left chest. Physics such as salts or castor oil were taken every six to eight weeks. He described the sensation of fullness during the first attack "like brickbat in the side, one-half brick high and one brick sideways (lateromedially)," clearing with slight change in position, thereby relieving pain and permitting him to recline. This sensation upon change of position occurred throughout his first attack.

The second attack, in 1941, was of one week's duration. While shucking corn, pain appeared in the same location, but migrated to the left sacral area. Pain was characterized as more continuous and less severe, but was still stabbing in character and required bed confinement. Pain was aggravated in attempting to get out of bed.

The third attack occurred in 1948. While sitting in church he was seized with a sharp, hard pleurisy pain under the left shoulder blade, passing through to the front of the chest. It was worse upon deep breathing. Rubbing liniment was prescribed, and the pain shifted to the location originally described in first attack, left anterior axillary line midway between the left costal border and left iliac crest, and situated moderately deep. After the shift in location of pain, some relief was experienced. At this point, the patient described "vibration and blubbering" like fluid in the left chest, changing position with posture and motion. Later on, edema of the feet and ankles was noticed, and a roentgenogram of his chest was advised.

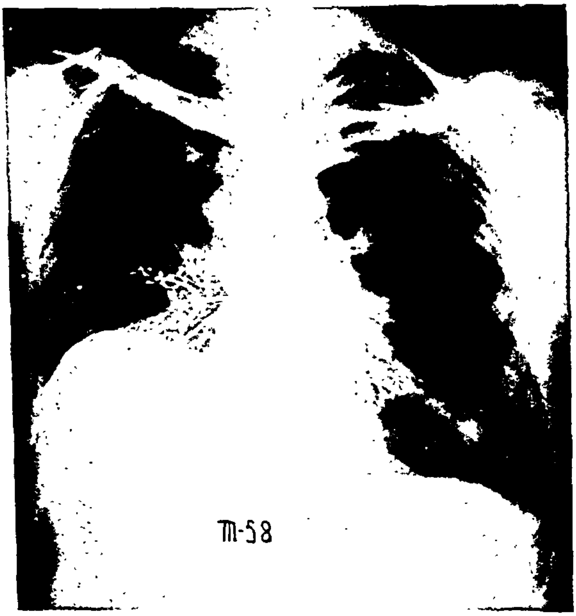

Fig. 1.—Bronchogram shows extrapleural density in left chest displacing bronchi.

Past history is negative for any serious illnesses, accidents, or operations. In his family history, his mother had died in 1904 with tuberculosis.

Positive findings of physical examination consisted of: left pupil slightly irregular; nasal septum deviated to the left with partial obstruction; thorax symmetrical with restricted respiratory movements, along the right margin; the right lung exhibited increased breath sounds; the left lung presented dullness at the base, especially anteriorly, with absent breath sounds and absent fremitus at the base anteriorly; axillary fremitus was heard on the left side.

Laboratory findings were: W.B.C. 10,900; juveniles 1 per cent, stabs 1 per cent, segmenters 80 per cent, small and large lymphocytes 18 per cent. Sedimentation rate 10 mm.

(Cutler method). Urinary findings were negative. R.B.C. 4,000,000, with 76 per cent Hb. (Sahli). Blood type A. Aspirated bronchial secretions were cytologically negative for neoplastic cells.

*X-Ray Findings.*—Bronchogram (Fig. 1) demonstrated a dense mass in the left lower chest reaching the level of the ninth rib and displacing the left lower bronchi superiorly and the heart slightly to right. Fig. 2 is a radiograph taken on the sixth postoperative day. The diaphragm is outlined. A small quantity of fluid is present.

*Operative Report.*—A large, rounded, well-encapsulated mass was located on the left diaphragm with major fibrous attachment in the form of a short pedicle located posterocentrally in an area about 4 by 3 cm. and which mass, when separated, left bleeding surfaces on both diaphragm and tumor capsule. Pleural adhesions were present laterally and medially connecting to diaphragm and to mediastinum. There were large adhesions connecting the tumor firmly to the inferior aspects of the lung which appeared and felt atelectatic. The tumor was well demarcated and encapsulated. Recovery was uneventful. The patient was discharged from the hospital on the sixth postoperative day.

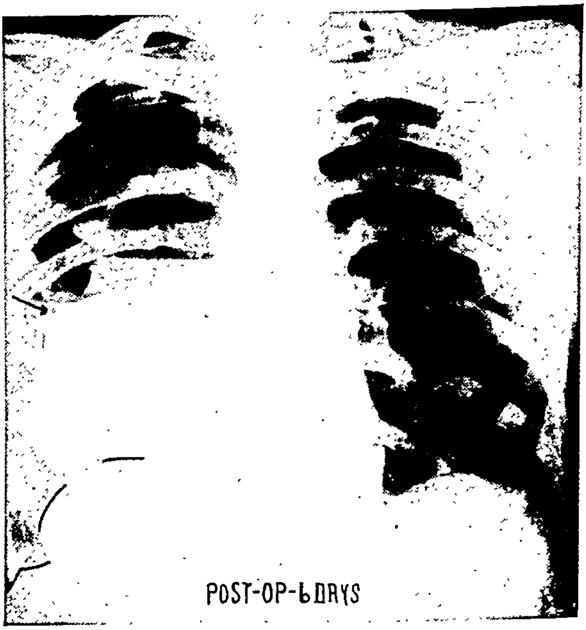

Fig. 2.—Radiograph taken six days after removal of tumor shows outline of left diaphragm, small quantity of fluid, lung re-expanding.

*Gross Pathology Report.*—Tumor mass (Fig. 3) measured 15 by 10 by 10 cm. and weighed 190 grams. Surface was smooth except for areas of pleural and diaphragmatic attachment and a bosselated area approximately 4 cm. in diameter. Consistency was moderately firm and resilient and cut surface exhibited bulging with occasional yellowish degenerated areas, tumor being similar in gross characteristics to uterine leiomyomas. It was circumscribed and encapsulated.

*Microscopic Description.*—(See Figs. 4 and 5.) Section showed tumor composed of solid masses of spindle cells. There was a moderate amount of intercellular substance. The nuclei were very numerous. They were of moderate size and of uniform staining. The chromatin was evenly dispersed. Mitoses were rare. There was no special architectural arrangement beyond tendency of cells to form thick bundles. These cells did not appear especially active.

TABLE I

| MALIGNANT | | BENIGN | |
|---|---|---|---|
| *Sarcoma:* | | | |
| Fibrosarcoma[1-5] (present report) | 6 | *Lipoma:*[20, 21, 22, 23] | 4 |
| Fibromyosarcoma[6] | 1 | *Fibroma:* | |
| Neurofibrosarcoma[7] | 2 | Fibroma[24] | 1 |
| Rhabdomyosarcoma[8, 9] | 2 | Fibromyoma[25, 26] | 2 |
| Leiomyosarcoma[10] | 1 | Rhabdomyofibroma[27] | 1 |
| Myosarcoma[11] | 1 | Angiofibroma[28] | 1 |
| Undifferentiated Myosarcoma[10] | 1 | Neurofibroma[29] | 1 |
| Endothelial Sarcoma[12] | 1 | | |
| Round Cell Sarcoma[13] | 1 | *Lymphangioma:*[30] | 1 |
| Mixed Cell Sarcoma[14] | 1 | | |
| Sarcoma[15] | 1 | *Chondroma:*[31] | 1 |
| Undifferentiated Sarcoma[16] | 1 | *Dermoid cyst:*[32] | 1 |
| *Endothelioma:* | | *Cystic tumor:*[33, 34, 35] | 3 |
| Fibroangioendothelioma[17] | 1 | | |
| Hemangioendothelioma[18] | 1 | | |
| Mesothelioma[19] | 1 | | |
| Total | 22 | Total | 16 |

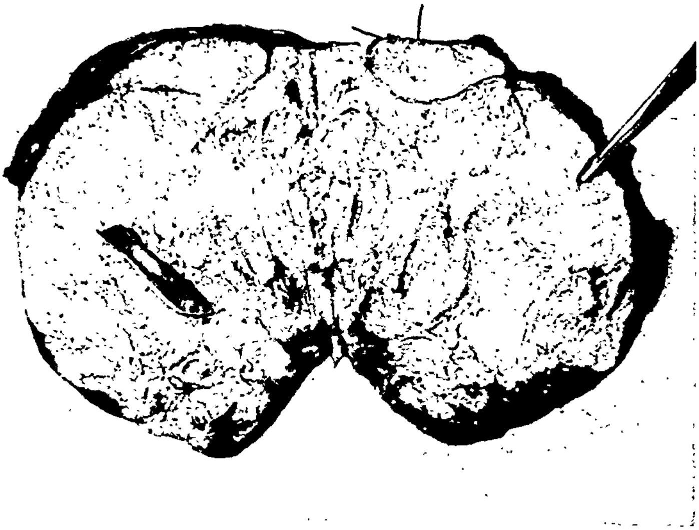

Fig. 3.—Hemisection of tumor. Blade points to yellowish degenerated area.

The tissue appeared to be benign. Other portions of tissue were somewhat distorted. One area contained many capillary-sized spaces formed of cells similar to those of tumor, and here the fibrous nature of the tumor was not so marked.

*Subsequent Course.*—The patient remained symptom-free, with apparent recovery, for one year. Then symptoms began to recur, finally leading to biopsy of recurrent tumor mass in June, 1950. This tumor was seen to be larger than a cocoanut, of softer consistency, red in color, with increased vascularity as compared to the first tumor. Attachment was extensive and confined within the medial half of the diaphragm with definite invasion of that structure, whereas the original tumor did not invade the diaphragm. The microscopic picture (Fig. 6) was also different with varying degrees of fibrosarcomatous activity in various areas. There were no areas as inactive and benign in appearance as was the original tumor. It was considered inoperable on exploration. A biopsy showed the tumor to be a malignant fibrosarcoma. Following deep x-ray therapy, the size of the tumor was not greatly altered, but the patient apparently was more comfortable. Subsequent radiograph (Fig. 7) showed the recurrence of the diaphragmatic tumor mass, with metastases to both lungs. The patient died thirty-four months after removal of the initial tumor.

## COMMENT

It is perhaps idle to speculate whether these were two separate tumors, or the second one a recurrence, with malignant degeneration of the original tumor. We might assume the original tumor to be a histologically and ap-

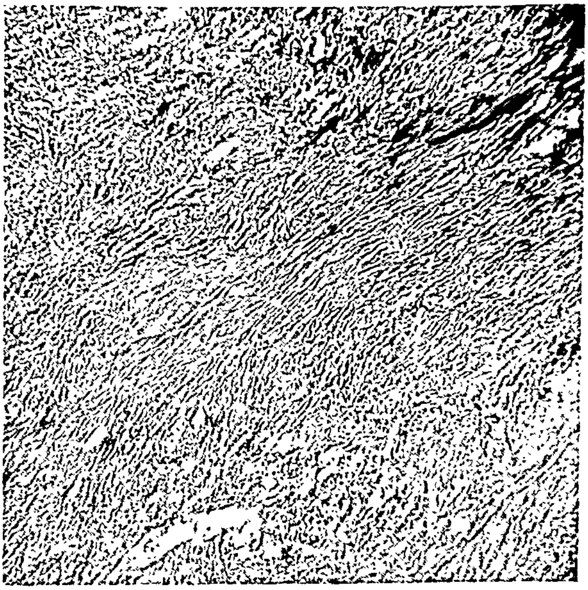

Fig. 4.

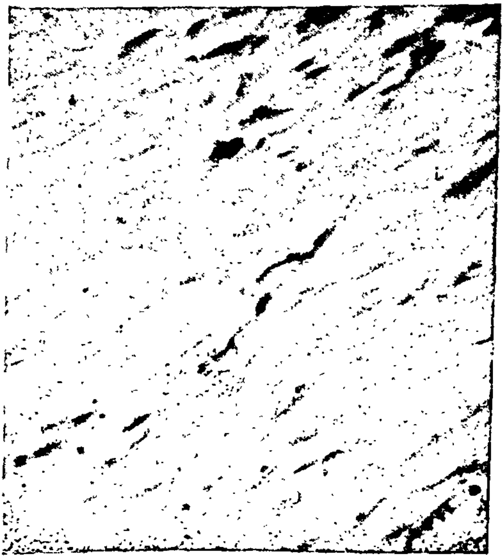

Fig. 5.

Figs. 4 and 5.—Photomicrographs showing low (×60) and high (×530) magnification, the inactive phase.

parently benign leiomyofibroma of the diaphragm which underwent malignant anaplasia and degeneration in its recurrence in the form of fibrosarcoma. The original tumor, however, was probably a low-grade fibrosarcoma, which became anaplastic and activated in its recurrence.

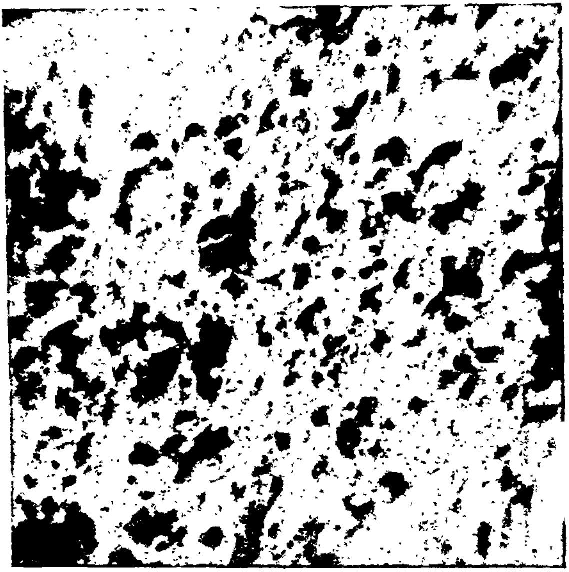

Fig. 6.—Photomicrograph (×530) demonstrating marked change and fibrosarcomatous activity of the recurring tumor.

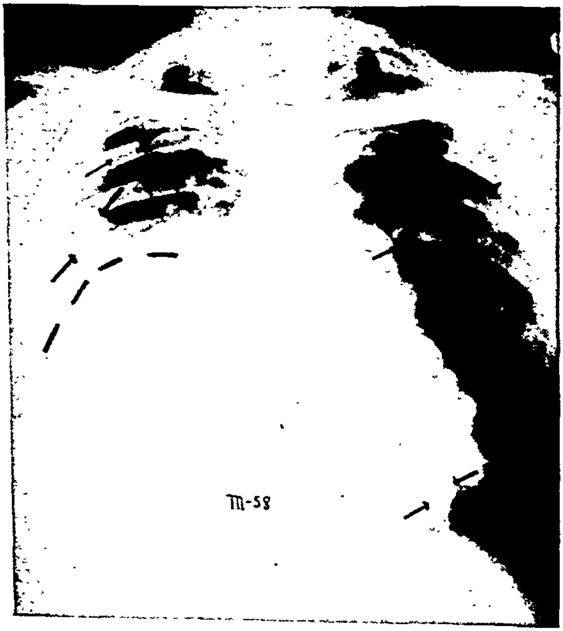

Fig. 7.—Radiograph thirty-three months after removal of initial tumor, showing the recurrence in the diaphragm with metastases.

## SUMMARY

1. A case of fibrosarcoma of the diaphragm is presented.
2. A review of the literature revealed thirty-eight primary tumors which have occurred in this anatomic location.

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# THE REPAIR OF EXPERIMENTAL TRACHEAL DEFECTS WITH FRESH AND PRESERVED HOMOLOGOUS TRACHEAL GRAFTS

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IN 1940 Taffel[1] described the regenerative capacity of the trachea and several reports have appeared subsequently in the literature concerning experimental resection of the trachea in dogs.[2-7] A few reports of attempts to restore the continuity of the human trachea have been recorded.[8-10]

It is conceivable that situations that make bridging of defects in the trachea desirable may arise in dealing with neoplasms, inflammatory stenosis, or trauma in the cervical or thoracic trachea. One difficulty complicating anastomosis of the larger respiratory passages is the frequent development of strictures postoperatively. At this writing no reports describing the use of tracheal grafts to repair large tracheal defects could be found in the available literature.

Preliminary experiments were performed on the cervical trachea of anesthetized dogs in order to compare various methods of preservation and insertion of tracheal grafts. All tracheal grafts were obtained by clean, but not strictly aseptic, technique. One series of tracheal grafts were preserved in Tyrode's solution by a method similar to that reported by Gross[11] for the preservation of blood vessels. A second group of tracheal grafts were preserved by rapid freezing in a bath of methyl alcohol and dry ice and kept at a temperature of 70° C. below zero. A third group of tracheal grafts were preserved in a 4 per cent solution of formaldehyde. A segment of cervical trachea containing three to six cartilage rings was excised, and the resulting defect bridged with a homologous tracheal graft. This graft was fixed in position with interrupted No. 40 cotton sutures. An attempt was made to use a graft which approximated as nearly as possible the diameter of the excised segment of trachea. The graft was usually one to two rings longer than the section of trachea removed. All animals in this and in the following studies received 400,000 units of penicillin daily for five days postoperatively In this preliminary group of tracheal grafting experiments the results, shown in Table I, were the same whether the grafts were freshly obtained, frozen, or preserved in Tyrode's solution. Within a period of one to three weeks the grafted segment became narrowed to the point of complete obstruction.

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From The Experimental Surgery Laboratories, University of California School of Medicine.

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TABLE I. TRACHEAL GRAFTING WITHOUT SUPPORTING TUBE

| METHOD OF PRESERVATION | AGE OF GRAFT | LENGTH OF GRAFT | SURVIVAL TIME | RESULTS |
|---|---|---|---|---|
| Tyrode | 1 week | 4 rings | 22 days | Expired |
| | 2 weeks | 4 rings | 3 days | Obstructed* |
| | 10 days | 6 rings | 24 days | Obstructed* |
| | 5 days | 4 rings | 23 days | Obstructed* |
| | 4 days | 6 rings | 14 days | Obstructed* |
| Fresh | – | 6 rings | 11 days | Obstructed* |
| Frozen | 11 days | 4 rings | 11 days | Expired |
| | 2 weeks | 4 rings | 3 days | Obstructed* |

*Reoperated.

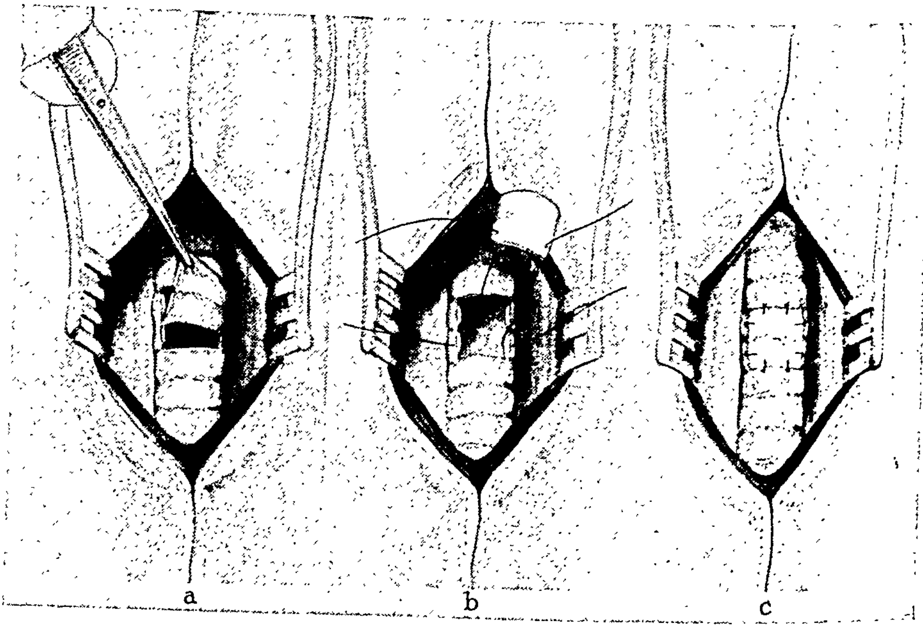


Fig. 1.—Creation of tracheal window and insertion of tracheal graft.

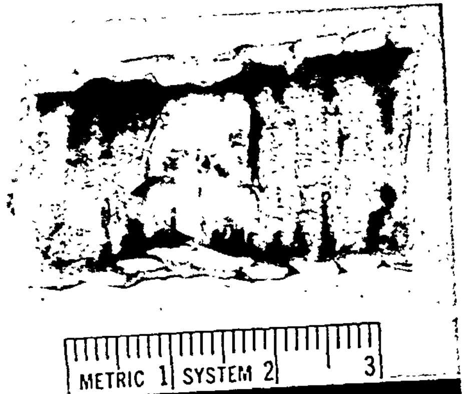
Microscopic examination of these specimens showed disintegration of the cartilage and replacement with fibrous tissue. The epithelial lining had disappeared, and the lumen of the transplanted graft was lined instead with chronic granulation tissue. The grafts preserved in formaldehyde were sloughed into the lumen of the trachea and were surrounded by a capsule of fibrous and granulation tissue. These grafts showed no evidence of being incorporated into the normal trachea.

From this group of experiments it appeared that, regardless of the method of preservation of the graft, postoperative stenosis developed in all instances in which no tracheal support was used.

In a second group of two animals a window was made in the anterior surface of the trachea. This defect involved two rings and one-third of the circumference of the trachea. A tracheal graft of comparable size and preserved in serum enriched Tyrode's solution was sutured into the area with interrupted No. 40 cotton (Fig. 1). No intratracheal supportive tubes were used in these dogs.

At the time of autopsy, four and one-half months after operation, these grafts were completely incorporated into the trachea (Fig. 2). The cartilage had survived, and the inner surface of the graft was covered by epithelium (Fig. 3).

Fig. 2.



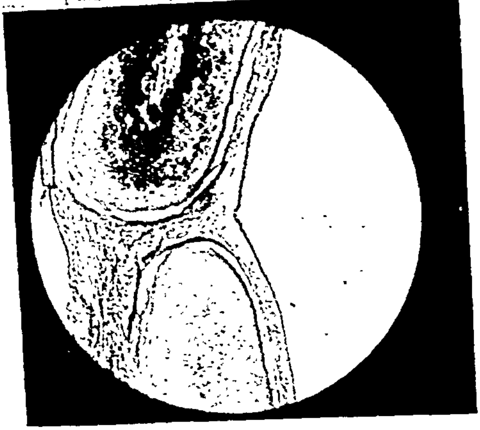


Fig. 3.

Fig. 2.—Gross appearance of tracheal window graft four and one-half months after operation.

Fig. 3.—Microscopic appearance of tracheal window graft four and one-half months after operation.

In a third group of experiments a method was developed in which an acrylic ("lucite") tube was used to splint the tracheal grafts inserted into the cervical and thoracic parts of the trachea.

The cervical portion of the trachea was exposed by a midline vertical incision and a segment of three to four rings of trachea excised. Into this defect a tracheal graft containing the specially designed acrylic tube was fixed in position with cotton sutures and No. 32 stainless steel wires, as indicated in

Figs. 4, 5, and 6. It was not necessary to use an intratracheal catheter during the grafting in the cervical trachea. These tracheal grafts were preserved in serum enriched Tyrode's solution for one to thirty days, and then implanted in seven dogs on eight occasions. In the first four animals the tube was fixed in position with No. 32 gauge, stainless steel wire, and became dislodged three to seven weeks later. A heavier, No. 24 gauge wire maintained the tubes in place in the last four successful cervical grafts. Three of these animals were alive and without respiratory symptoms after seven months of observation (see Table III).

Autopsy examination of the one animal sacrificed six months after tracheal grafting showed normal lung parenchyma. The acrylic tube remained securely in place (Figs. 7 and 8). The grafted segment showed cartilage remaining in its wall and complete regeneration of the lining by squamous epithelium (Fig. 9).

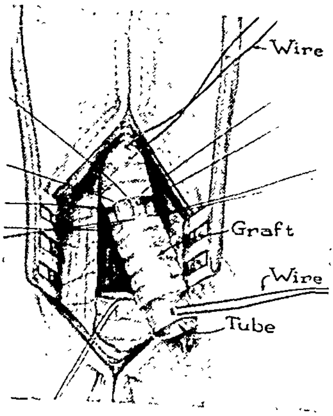


Fig. 4.

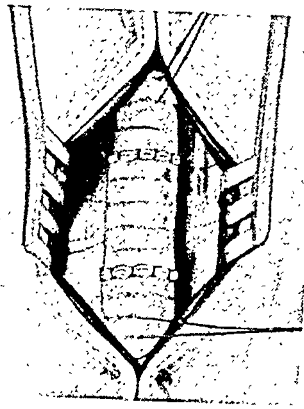


Fig. 5.

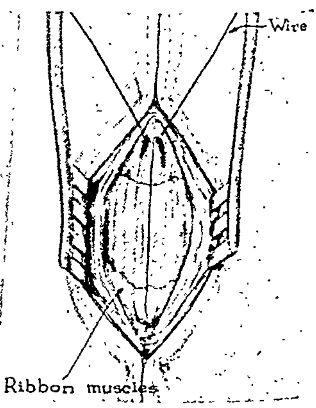


Fig. 6.

Fig. 4.—Insertion of graft containing acrylic tube.
Fig. 5.—Completion of suture lines.
Fig. 6.—Stainless steel wires passed through ribbon muscles and tied.

TABLE II. GRAFTING OF TRACHEAL WINDOWS WITH PRESERVED TRACHEAL GRAFTS WITHOUT SUPPORTIVE TUBES

| METHOD OF PRESERVATION | AGE OF GRAFT | LENGTH OF GRAFT | SURVIVAL TIME | RESULTS |
|---|---|---|---|---|
| Serum enriched | 2 days | 1 by 1.5 cm. | 4½ months | Sacrificed |
| Tyrode's solution | 4 days | 1 by 1.5 cm. | 4½ months | Sacrificed |

TABLE III. CERVICAL TRACHEAL GRAFTS USING ACRYLIC TUBE SUPPORT

| AGE OF GRAFT | LENGTH OF GRAFT | SURVIVAL PERIOD | RESULTS |
|---|---|---|---|
| 1 day | 10 rings | 20 days | Expired |
| 1 week | 8 rings | 49 days | Obstructed* |
| 12 days | 6 rings | 30 days | Expired |
| 1 day | 6 rings | 49 days | Expired |
| 1 day | 6 rings | 6 months | Sacrificed |
| 1 mo. | 6 rings | 7 months | Living |
| 2 days | 6 rings | 7 months | Living |
| 15 days | 6 rings | 7 months | Living |

*Reoperated.

TABLE IV. THORACIC TRACHEAL GRAFTS USING ACRYLIC TUBE SUPPORT

| AGE OF GRAFT | LENGTH OF GRAFT | SURVIVAL PERIOD | RESULTS |
|---|---|---|---|
| 4 days | 5 rings | 10½ months | Sacrificed |
| 8 days | 5 rings | 10 months | Living |
| 15 days | 5 rings | 8 months | Sacrificed |
| 21 days | 5 rings | 10 months | Sacrificed |
| 10 days | 5 rings | 10 months | Living |
| 7 days | 6 rings | 8½ months | Living |
| Fresh | 6 rings | 5 months | Sacrificed |
| 1 day | 6 rings | 5 months | Sacrificed |
| Fresh | 6 rings | 8 months | Living |
| 14 days | 6 rings | 3¼ months | Sacrificed |

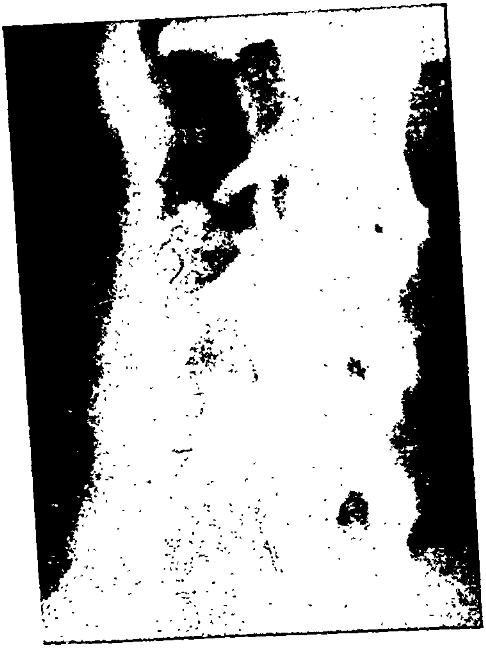
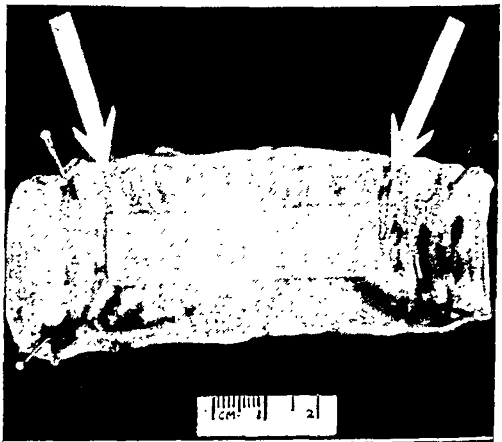


Fig. 7.—Roentgenogram of cervical trachea showing intratracheal acrylic tube in place.

In a fourth group of animals the thoracic trachea was exposed by an incision developed through the right fourth intercostal space. The right vagus nerve was injected with 2 c.c. of 4 per cent solution of procaine to obviate vagal reflexes. When the trachea was incised a sterile intratracheal catheter was placed into the distal end and secured by a suture of No. 40 cotton (Fig. 10). Controlled respirations were maintained through this catheter. A segment of the intrathoracic trachea 3 to 4 rings in length was excised, and a graft containing an acrylic tube was sutured in place in a manner similar to that described for the grafting of the cervical trachea. After the proximal suture line was secured, the intratracheal catheter was withdrawn. The distal sutures were then rapidly drawn up and tied, and respiratory exchange was maintained by means of an oral intratracheal catheter. The wire sutures were passed through the paravertebral musculature to prevent their erosion through the tracheal cartilage (Fig. 11). Air was withdrawn from the pleural space by a rubber catheter, and the thorax closed without drainage. At the close of the procedure 5 c.c. of a solution containing 40,000 units of penicillin per cubic centimeter were instilled locally into the pleural space.

Fig. 8.



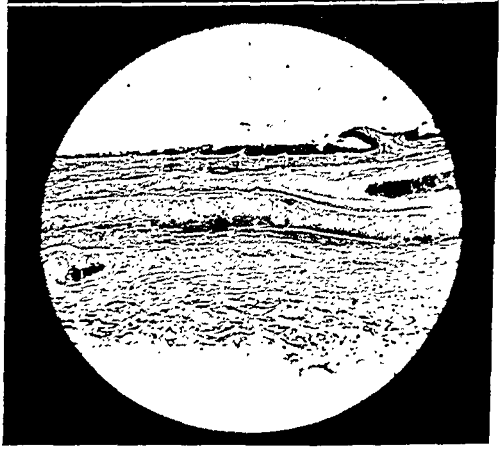


Fig. 9.

Fig. 8.—Gross specimen of grafted segment of trachea containing acrylic tube.

Fig. 9.—Microscopic section (×17.2) showing regenerated epithelial lining and cartilage remaining in cervical graft. Sacrificed six months after insertion of graft.

Ten animals had segments of thoracic trachea removed and replaced with tracheal grafts preserved in serum-enriched Tyrode's solution. These grafts were preserved from one to three weeks before they were used. The freshly obtained tracheal grafts were also used in this group of experiments. All of the dogs survived this operation, and have been observed for periods up to ten and one-half months postoperatively (See Table IV).

It was found that the tube could be removed readily by means of the bronchoscope, when the wires were broken or cut at a second-stage operation. The fine, No. 32 gauge wire was used in all of these animals.

In four of the six animals sacrificed at varying intervals, the wire had disrupted and the tube was dislodged. Autopsy examination revealed normal lung parenchyma. The graft was shortened and the lumen narrowed to about two-thirds of the normal diameter. One animal apparently had ejected the tube, as it was absent at autopsy. On microscopic examination of the grafted areas some chronic inflammatory reaction was observed; however, the grafts

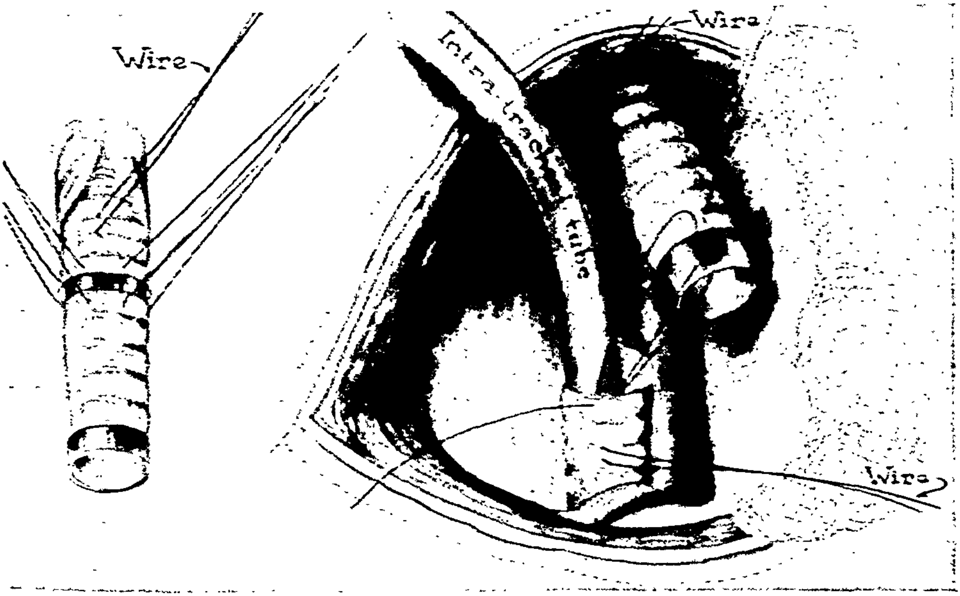


Fig. 10.—Sterile intratracheal catheter in place and graft being sutured into the trachea.

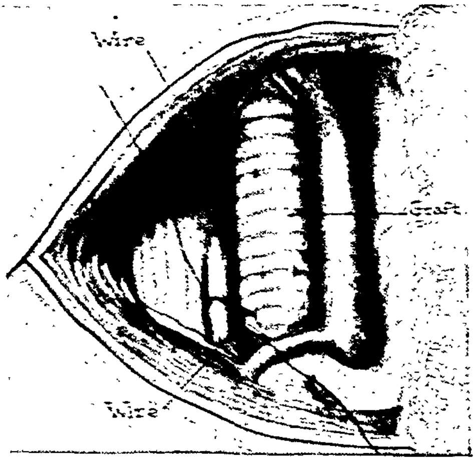


Fig. 11.—Completion of suture lines.

TABLE V. PNEUMONECTOMY AND TRACHEAL GRAFT USING ACRYLIC TUBE

| GRAFT AGE | SURVIVAL PERIOD | RESULTS |
|---|---|---|
| 1 week | 8 hours | Expired |
| 3 days | 6 hours | Expired |
| Fresh | 1 week | Expired |
| 5 days | 3 months | Expired |
| 4 days | 7 months | Living |

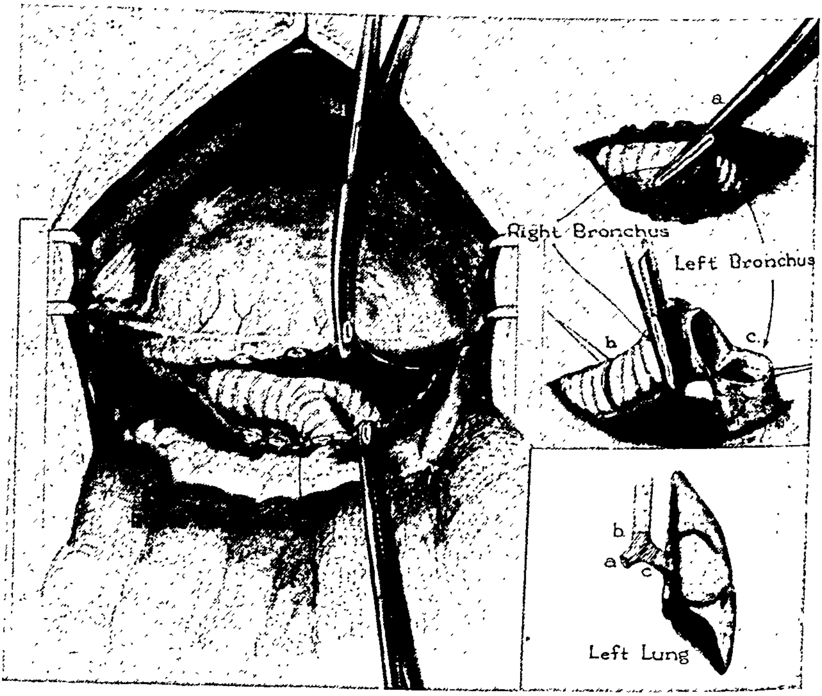


Fig. 12.—The right lung removed and the carina and a portion of the left main-stem bronchus to be removed.

were lined with squamous epithelium. Some cartilage remained in the area of the graft in all of these specimens.

In a fifth group consisting of five animals a right pneumonectomy was performed through an incision in the right fifth intercostal space. After removal of the right lung, the distal 3 rings of trachea, the carina, and first 2 rings of the main-stem bronchus were freed from the surrounding fascia and excised (Fig. 12). A free, homologous tracheal-carinal graft preserved in serum-enriched Tyrode's solution was then inserted into this defect by a technique similar to that already described for thoracic tracheal grafting (Fig. 13). The right main-stem bronchus on the graft had been closed with interrupted mattress sutures of No. 40 cotton and reinforcing continuous suture of No. 40 cotton. The acrylic tube used to splint this defect was slightly angulated and tapered to allow for the difference in size of the tracheal and bronchial stumps.

The first two animals in this group died six and eight hours postoperatively from leakage of the anastomosis and pneumothorax. One animal died one week postoperatively with disruption of the graft. One animal expired three months after operation, having developed a pneumonitis following a

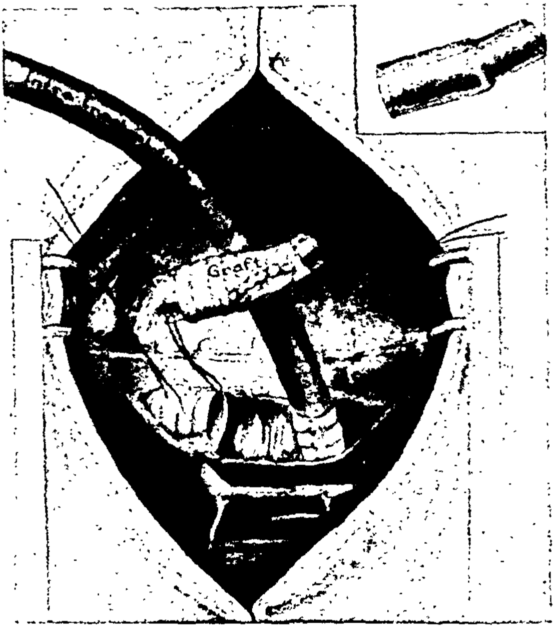


Fig. 13.—Tracheal carinal graft being inserted.

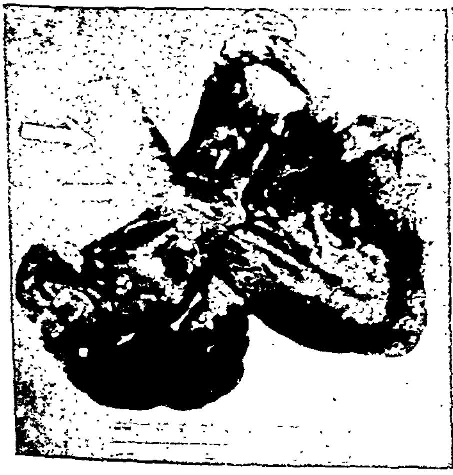


Fig. 14.

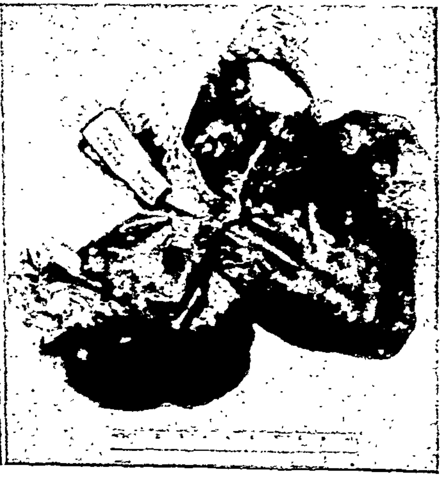


Fig. 15.

Fig. 14.—Specimen of left lung and trachea containing tracheal graft and acrylic tube.
Fig. 15.—Specimen of left lung and trachea containing tracheal graft and acrylic tube (specimen opened).

fight with another dog. Pathologic examination revealed the acrylic tube in place with well-defined cartilage remaining in the tracheal carinal graft (Figs. 14 and 15). There was pneumonitis and consolidation in the lower and middle lung fields. One animal remains living and apparently well seven months postoperatively (Table V).

## SUMMARY

Preserved homologous tracheal window grafts have been successfully implanted into the trachea of two animals.

Complete homologous tracheal grafts have been implanted into the cervical and thoracic trachea in twenty-eight dogs with no operative mortality. In ten animals in which no means of internal support was used, the grafts became stenosed within one to three weeks after implantation. When cervical and thoracic homologous tracheal grafts were supported by an acrylic tube and held in position with stainless steel wire of appropriate size, the tubes remained in place for the duration of the experiment and caused no apparent difficulty. Cartilage has remained in these grafts for at least ten and one-half months, and the epithelial lining has been completely replaced by squamous epithelium during this period.

A method has been described for removing one lung, the distal trachea, carina, and a portion of the left main-stem bronchus of experimental animals, and bridging this defect with a tracheal graft and supporting acrylic tube.

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# HODGKIN'S DISEASE OF THE LUNG WITH CAVITATION

## REPORT OF THREE CASES

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HODGKIN'S disease is characterized by a marked involvement of lymphoid and reticulo-endothelial tissue. The location of the disease within the chest will therefore be determined by the distribution of these tissues, which are present in abundance in the interstitial tissue of the lung and the mediastinum.

The disease can originate in the lung itself, or the lung can be involved by direct invasion from affected glands in the mediastinum. The further spread of the process within the lung may follow the peribronchial or perivascular lymph vessels, and can result in peribronchitis or intrabronchial changes with granulations (Moolten[17]). Where the granulations and thickening of the bronchial wall are extensive, they may cause bronchostenosis, and the findings will be indistinguishable from a case of bronchiogenic carcinoma (Higginson[11]). Another form of spread within the lung is the more peripheral involvement of the alveolar wall, with the production of granulations followed by consolidation of lung segments or lobes resembling an ordinary pneumonia or pulmonary tuberculosis. A less common manifestation is the occurrence of well-circumscribed isolated nodules in the lung, giving a picture similar to secondaries in the lung. A miliary form has been described by Vieta and Craver.[3]

Central necrosis and the formation of cavities are not met with in Hodgkin's disease in other parts of the body, but cavitation does occasionally occur in the lungs, probably as a result of the special anatomy of the pulmonary tissue. When a cavity has been formed within the infiltration, pulmonary tuberculosis, lung abscess, or abscess in combination with a bronchiogenic carcinoma have to be considered in the differential diagnosis.

The symptoms caused by pulmonary Hodgkin's disease are more or less the same as those met with in the more common pulmonary diseases. They are fever, cough, night sweats, pain in the chest, loss of weight, and dyspnea. If a cavity is present the patient will probably cough up purulent sputum; hemoptysis is not common, but has been recorded. Changes may take place in the peripheral blood, and patients suffering from Hodgkin's disease may have a normal, high, or low, blood-cell count. The sedimentation rate is generally elevated. A positive diazo reaction in urine is suggestive, but not necessarily present. In about 10 per cent of the patients suffering from Hodgkin's disease one finds generalized pruritus which is a very troublesome complaint.

The prognosis in cases with pulmonary Hodgkin's disease is not as good as in patients afflicted with the same disease in other parts of the body. In the cases with pulmonary manifestations and cavitation reported in the literature, the survival from the time of the first symptom to death varies between two

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and nine years. The presence of constitutional symptoms is often met with in patients with pulmonary involvement, and is a bad prognostic sign.

During the last two years, three patients suffering from pulmonary Hodgkin's disease, with cavity formation, have been observed in our department.

Case 1.—A. E., a woman, aged 30 years, previously had had no illness. In the autumn of 1947 she was vaccinated with B.C.G., but the Pirquet and Mantoux tests were still negative in May, 1948. In December, 1948, she became ill with fever, pain in the right side of the chest, and felt listless. She developed an unproductive cough and her B.S.R. was 110 mm. The Pirquet test was still negative, but the Mantoux test was found to be positive. A radiograph of the chest showed an infiltration in the right upper lobe, and one month later a cavity had appeared in this opacity. In another hospital a diagnostic aspiration of the lung cavity was performed and purulent fluid obtained. The fluid was sterile, and on culture no growth of bacilli occurred. Because of the elevated temperature and her clinical appearance she was treated with systemic penicillin; this treatment was followed by a fall in the temperature and clinical improvement for a short time.

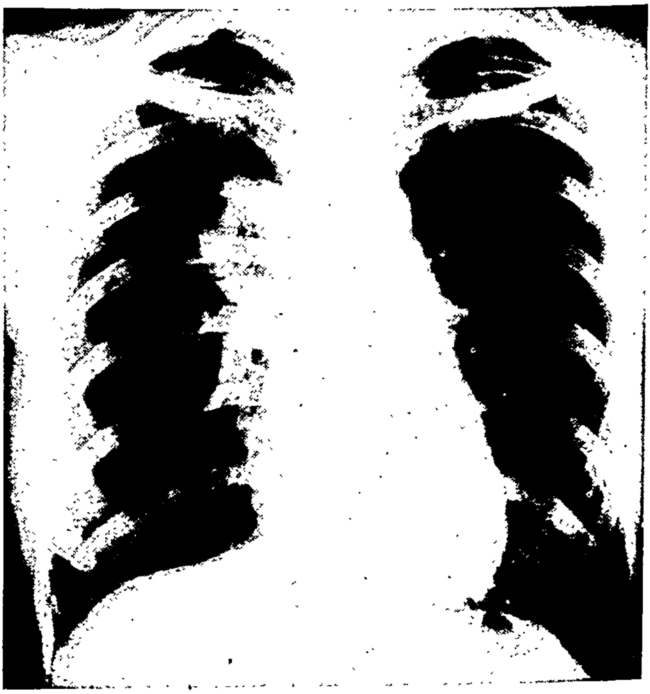

Fig. 1.—Anteroposterior radiograph showing a density in the right upper lobe with a cavity in Case 1.

In the beginning of the year 1949 she became ill again with chills, elevated temperature, and this time coughed up a mouthful of blood-stained purulent sputum. After this she improved, and her condition was thought to be caused by a lung abscess.

She was admitted to the Surgical Department A in February, 1949. Her general condition was fair, she had moderate anemia, B.S.R. was 109 mm. The white blood cell count was 8,200, with 15 per cent lymphocytes. Bronchoscopy was negative. The radiographs of the chest showed a density with a large cavity in the anterior segment of the right upper lobe (Figs. 1 and 2).

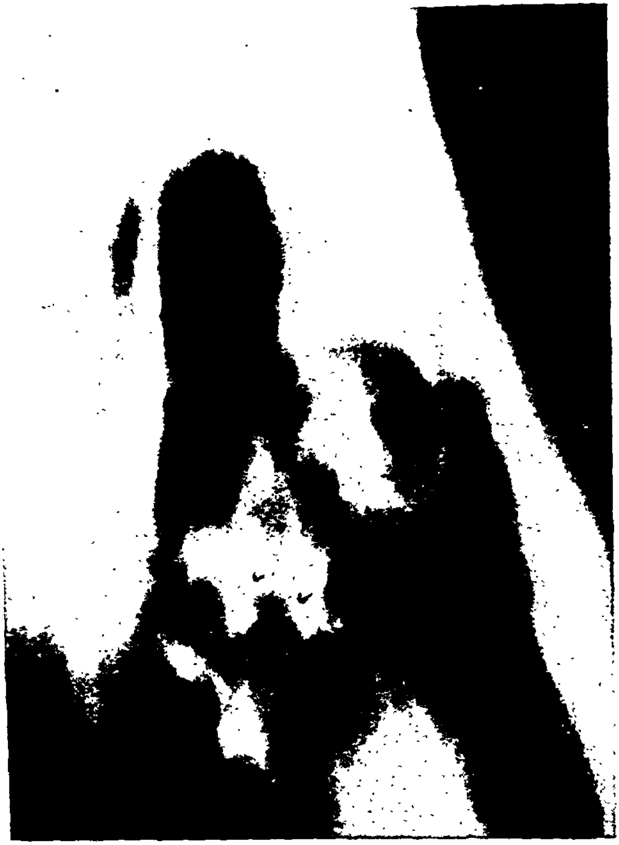

Fig. 2.—Lateral tomograph showing the cavity in the anterior segment of the upper lobe in Case 1.

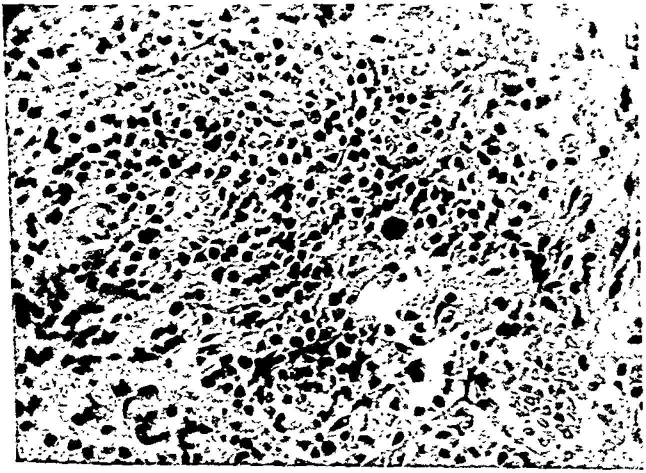

Fig. 3.—Photomicrograph of the involved lung in Case 1.

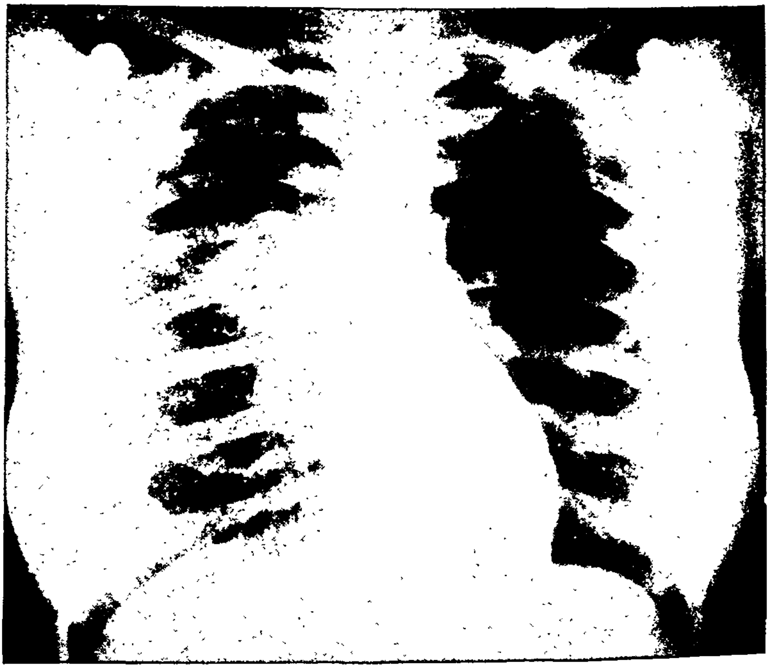

Fig. 4.—Anteroposterior radiograph showing a density in the right upper lobe with a cavity in Case 2.

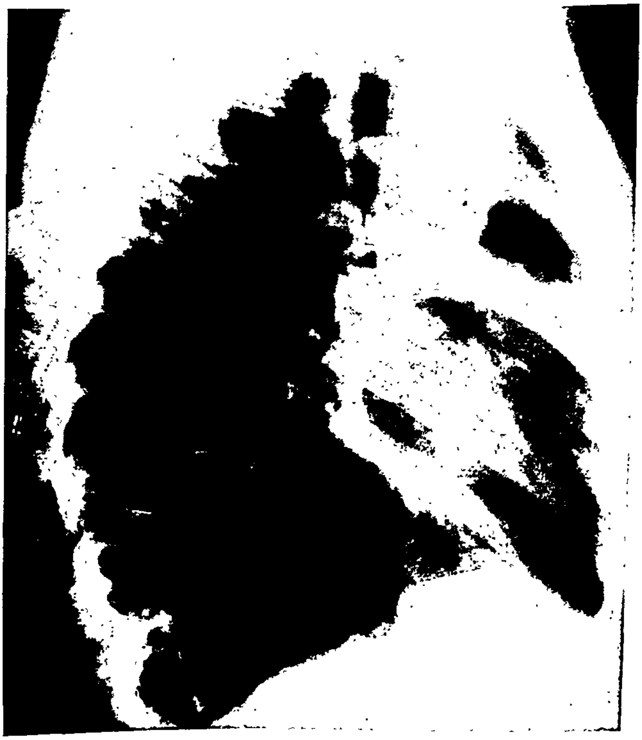

Fig. 5.—Lateral tomograph showing the cavity in the anterior segment of the right upper lobe in Case 2.

On March 2 a thoracotomy was performed on the right side. Firm adhesions were present between the chest wall and the anterior segment of the upper lobe, which was adherent to necrotic glands in the mediastinum. Along the internal mammary artery in the intercostal spaces, small fluctuating areas were found and were thought to be necrotic glands. A tuberculous process was considered the most likely diagnosis. The upper and middle lobe and the necrotic glands in the anterior mediastinum were removed.

The histologic examination of the lobes and the glands proved to be Hogkin's disease (Fig. 3).

The patient received postoperative radiotherapy, but later she developed a retrosternal fistula. She gradually lost ground, and died two years after the operation.

No post-mortem examination was obtained.

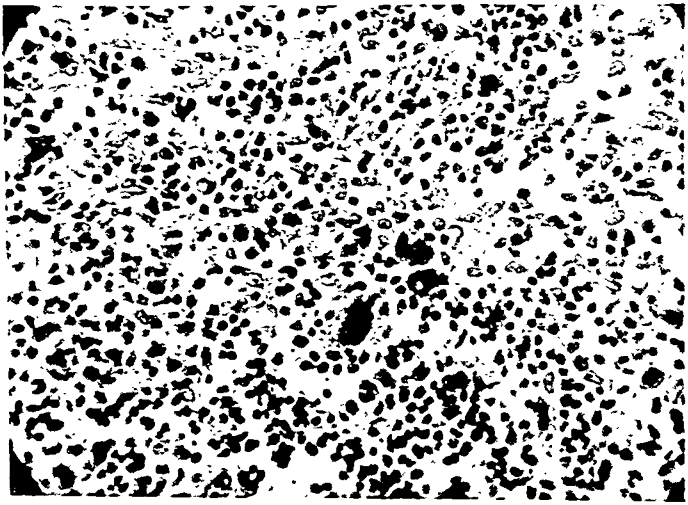

Fig. 6.—Photomicrograph of the involved lung in Case 2.

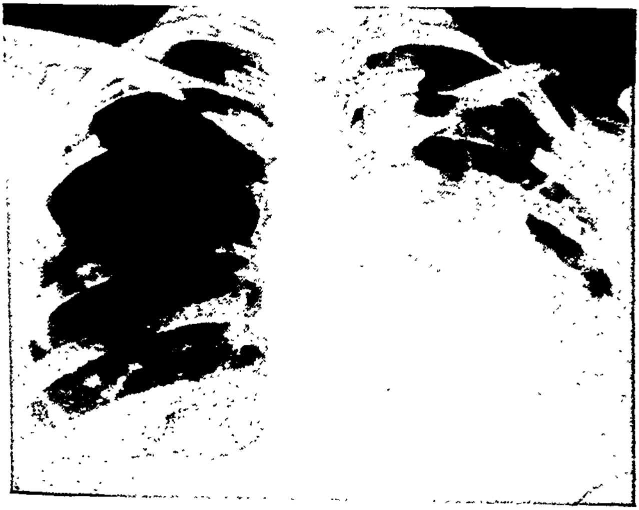

Fig. 7.—Anteroposterior radiograph showing infiltration of the opposite lung. The remaining lower lobe of the operated lung is clear.

CASE 2.—J. B., a woman, aged 21 years, who in 1948 had had jaundice of two weeks' duration. Otherwise she felt well, and there was no report of any previous illness. In the autumn of 1948 a routine mass radiograph revealed a density with cavitation in the anterior segment of the right upper lobe. She did not feel ill, but was admitted to a hospital for further investigation. The B.S.R. was 100 mm.

She was admitted to the Surgical Department A in February, 1949. Her general condition was good; there were no abnormal physical findings in her lungs. No glandular enlargement was palpated. She had a moderate anemia, B.S.R. was 66 mm., and the white blood cell count was 11,000, with 20 per cent lymphocytes. Bronchoscopy was negative. The radiographs of the chest showed a density in the anterior segment of the upper lobe with cavitation (Figs. 4 and 5).

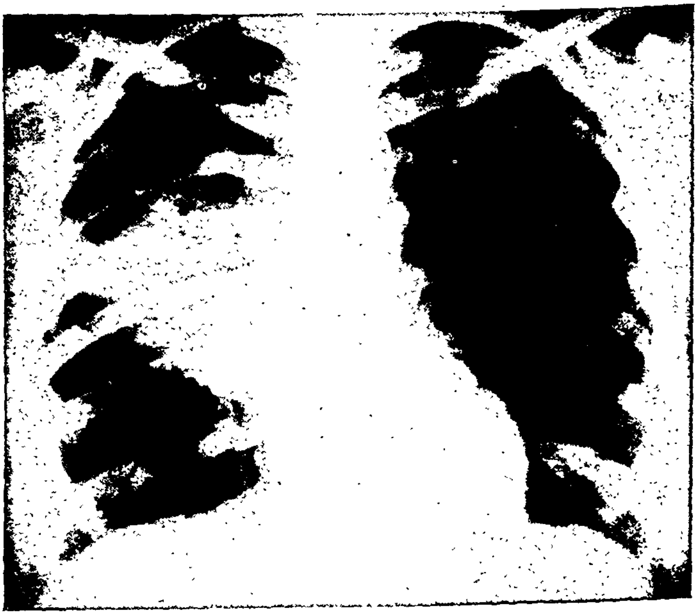

*Fig.* 8.—Anteroposterior radiograph showing a density in the right upper lobe with a cavity in Case 3.

On the diagnosis of lung abscess and the possibility of Hodgkin's disease, a thoracotomy was performed on March 4. A mass the size of a duck's egg was found in the anterior segment of the right upper lobe, which was adherent to the mediastinal pleura and the middle lobe. Some small glands along the upper lobe bronchus seemed to be involved. The upper and middle lobes were removed.

The histologic examination showed a polymorphous picture characteristic of Hodgkin's disease (Fig. 6).

She received postoperative radiotherapy to the mediastinum and to the operated side, but only five months later a shadow in the left hilum and one in the posterior part of the left lower lobe appeared. She was again treated with irradiation to the mediastinum and the left lung. In spite of this, the disease progressed on the left side, and her general condition deteriorated steadily. Two years later she had marked infiltration of the left lung (Fig. 7).

CASE 3.—I. H., a woman, aged 53 years, who previously had been healthy. A mass radiograph two years previously showed normal lungs and mediastinum. The Pirquet test was positive.

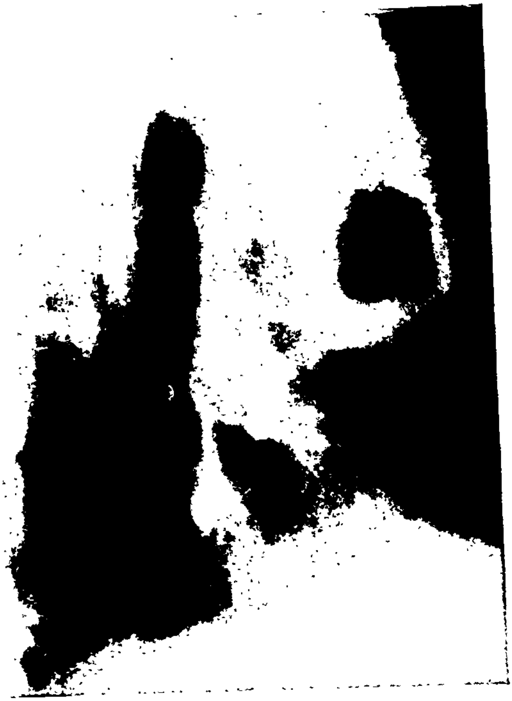

Fig. 9.—Lateral tomograph showing the cavity in the anterior segment of the upper lobe in Case 3.

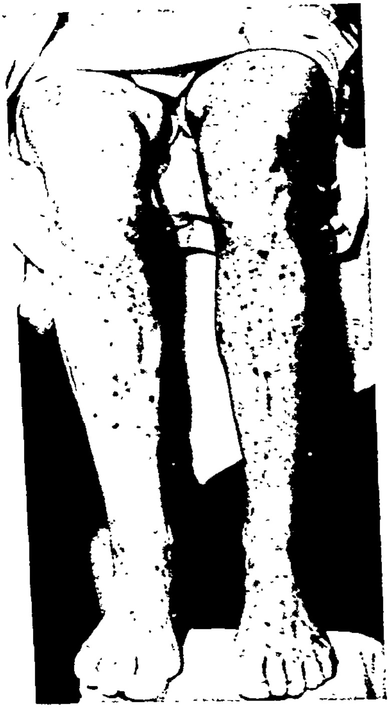

Fig. 10.—Photograph showing involvement of the skin in Case 3.

In December, 1950, she developed an exanthema of the trunk and the extremities with a severe and troublesome pruritus.

In January her B.S.R. was 100, and she was advised to have a roentgenogram of her chest made. This showed a density in the anterior segment of the right upper lobe with a cavity. (Figs. 8 and 9.) She was admitted to the hospital for further investigation. Her general condition was good but she complained of severe itching and her exanthema showed many scratch marks (Fig. 10). The temperature was normal, and the B.S.R. was 57 mm. The white blood count was 26,000. There was a marked anemia (73 per cent). The diazo reaction was positive.

She was transferred to the Surgical Department A on March 15, 1951, with a diagnosis of Hodgkin's disease.

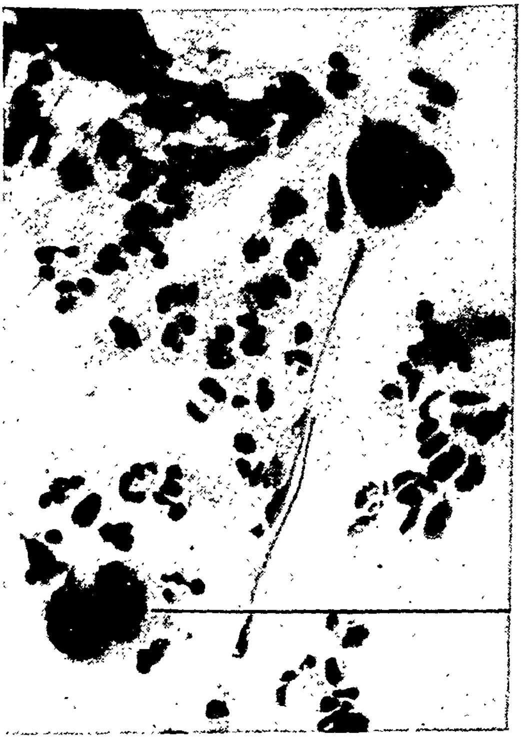

Fig. 11.—Photomicrograph of smear showing giant cells of the Reed-Sternberg type in Case 3.

Bronchoscopy showed some broadening of the carina, and from the right upper lobe bronchus reaccumulating purulent secretion was sucked out. A smear was examined and this showed giant cells very much like the Dorothy Reed-Sternberg cells (Fig. 11). A thoracotomy was performed on March 27, 1951, and a large mass was found in the anterior segment of the right upper lobe, and large glands in the mediastinum were densely adherent to the lung. The superior vena cava was embedded in the involved glands and firmly adherent. It was not considered possible to remove the glands radically, and only bits for section were taken.

The diagnosis was confirmed and proved to be Hodgkin's disease (Fig. 12). There was no evidence of tuberculosis. Postoperative irradiation resulted in some decrease in the pulmonary infiltration two months after operation.

## DISCUSSION

The three reported cases were all women aged 20 to 53 years. The symptoms referable to the respiratory tract prior to admission were very slight.

They all had a high B.S.R.—100 or more mm. in one hour. The pulmonary lesions in all three cases were localized to the anterior segment of the upper lobe on the right side. They all had adhesions to the mediastinal pleura, and in all cases enlarged retrosternal glands were found. In none of the cases was general lymphadenopathy present.

In pulmonary Hodgkin's disease it will always be a problem where the primary site of the lesion is, if the disease has started in the lung or in the mediastinum. This will be of considerable importance in deciding the justification for surgical treatment.

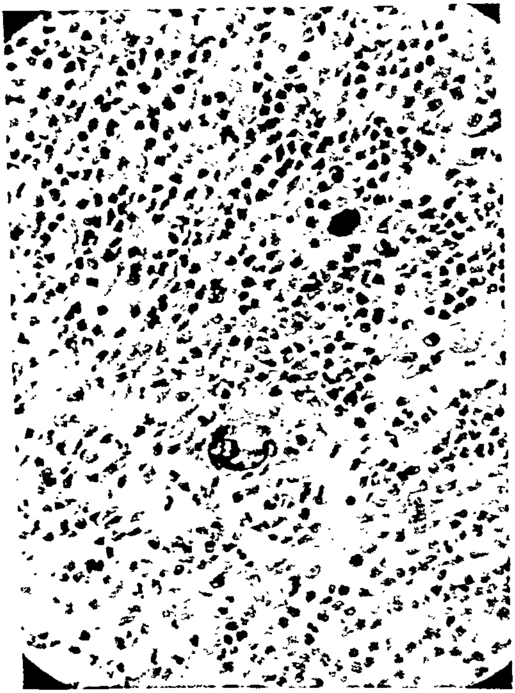

Fig. 12.—*Photomicrograph of the involved lung in Case 3.*

That the disease should have started in the lung and later extended to the glands in the mediastinum is not very likely, as the first group of lymph glands, situated along the larger bronchi, was involved in only one of our cases. The most likely sequence is that the disease in the lung has extended from involved glands in the mediastinum by direct extension through the pleura.

In the literature nine cases of Hodgkin's disease with cavitation in the lung have been recorded. The site of the process was the right lung except in one instance (Rubenfeld[19]). No clue, as to the primary site of the disease, was found in these cases.

A positive diazo reaction in the urine was suggestive of Hodgkin's disease in a patient with thoracic manifestations, as here described.

A bronchial smear examined for Reed-Sternberg cells can, as occurred in one of our cases, verify the diagnosis.

The treatment for pulmonary Hodgkin's disease will be as for Hodgkin's disease elsewhere: roentgen therapy, nitrogen mustard, or surgical removal of the localized focus.

If a cavity in the lung is present and it is possible to remove the involved glands in the mediastinum, a lobectomy or pneumonectomy followed by irradiation and possibly nitrogen mustard seems to be the most logical treatment.

If there are necrotic and secondarily infected glands in the mediastinum, as in Case 1, surgical treatment seems to be of little value, and in our patient only shortened the time of survival.

The prognosis with irradiation alone is in the first instant not too bad, and regression of infiltrations and disappearance of cavities have been reported.

In spite of the rarity of pulmonary Hodgkin's disease with cavitation, one ought to have it in mind in the differential diagnosis of the more common lesions in the lungs.

An effective histologic examination of the bronchial secretion may be of help in obtaining the correct diagnosis.

## SUMMARY

Three cases of pulmonary Hodgkin's disease with cavitation are recorded. In all, the anterior segment of the right upper lobe was the site of the lesion. In all the cases the retrosternal mediastinal glands were involved. In one case the disease spread postoperatively to the other lung.

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# BRONCHIAL ADENOMA

## A Case Report

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MALIGNANT change in an adenoma of the bronchus was first reported by Zamora and Schuster[1] in 1937. Since then many articles have appeared with confirmatory evidence as to the potentially malignant status of the so-called bronchial adenomas, but there still remains some confusion in the literature regarding the exact histogenesis and nature of this neoplasm. However, in view of the possible malignant status of the tumor, it seems that the only feasible therapy is radical extirpation of the tumor and surrounding normal tissue. Nevertheless, local treatment is still advocated by some bronchoscopists. In order to accentuate the malignant possibilities of the bronchial adenomas, Doty[2] has recently re-emphasized that they are mixed tumors, as originally observed by Womack and Graham,[3] and suggested that they be called "mixed tumor adenomas." The following case is reported to illustrate the malignant potentialities of a bronchial adenoma, and to demonstrate that radical therapy may not always succeed.

### CASE REPORT

C. H. H., a 26-year-old man, was first seen at the North Carolina Baptist Hospital on February 12, 1943. He was referred by his physician with the following note: "Onset of illness January 7, 1943, with high fever, nausea, vomiting, and pain in the left lower chest and left shoulder. He was started on one of the sulfonamides with marked improvement. However, in a few days the fever, nausea, and chest pain recurred, and he was admitted to the hospital. Left thoracentesis yielded 250 c.c. of blood tinged fluid, which was sterile to culture. A roentgenogram of the chest showed atelectasis of the left lower lobe with a shift of the mediastinum to the left. The patient improved after the thoracentesis, and was discharged from the hospital Feb. 5, 1943. He re-entered on February 9, with a temperature of 103° F., pain in the left lower chest, and cough productive of white frothy mucoid material. Chest roentgenogram still showed atelectasis of the left lower lobe." Additional history from the patient revealed that he had had an early morning cough, productive of small amounts of white sputum, and night sweats, for twelve months, with a 25-pound weight loss in this interval. Respiratory tract infections had been frequent every winter as far back as he could remember. Increasing shortness of breath, palpitation, and some clubbing of the fingers had been present for four or five years; this had been attributed to hay fever, which he had to a moderate degree. There was no history of exposure to or familial incidence of tuberculosis.

*Examination:* Temperature, 102.6° F., pulse rate, 104; respiratory rate, 24; blood pressure 115/78. The patient was a well-developed, acutely ill, young white man who was frequently stricken with paroxysms of coughing. The pharynx was moderately injected.

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The trachea deviated slightly to the left. The left lung below the fifth rib was dull to percussion, with absence of breath sounds and fremitus. The heart was shifted to the left. No other abnormalities were noted.

*Laboratory Data:* Urinalysis was negative. Hemoglobin 10 Gm., R.B.C. 4.8 million, W.B.C. 17,700, with 76 per cent polynuclearcytes. Sedimentation rate (Wintrobe, corrected) 49.5 mm. per hour. Kahn negative. Sputum negative for acid-fast organisms. A roentgenogram of the chest was reported as showing atelectasis of the left lower lobe. Bronchoscopy revealed a papillomatous growth at the orifice of the left lower lobe bronchus. A biopsy was taken and reported as follows: *Gross:* specimen consisted of a small fragment of tissue which measured approximately 1 cm. in diameter. *Microscopic:* Sections showed irregular islands of cells composed of epithelium of the basal type. Many cystic areas of degeneration were seen. The tissue for the most part was situated in a dense sclerotic fibrous tissue stroma; however, myxomatous areas were seen. *Diagnosis:* Basal-cell carcinoma of the bronchus. It was entirely possible that in this case an adenoma of the bronchus was being dealt with, and the material for study merely represented a basal portion of such a lesion.

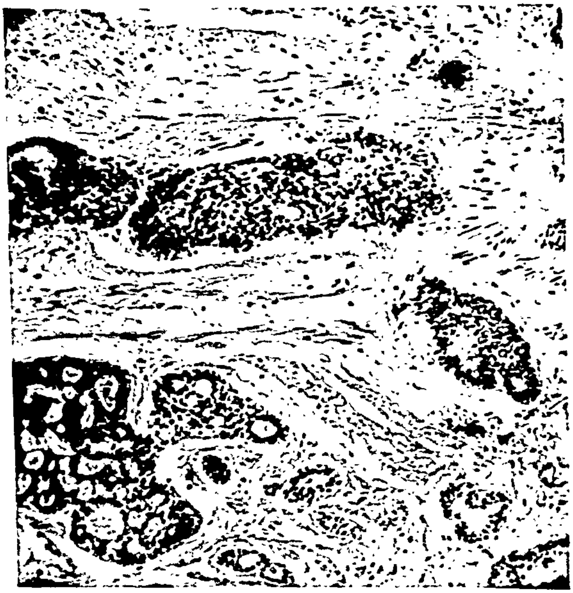

Fig. 1.—Photomicrograph of original biopsy specimen. (low power ×95), showing irregular epithelial glandular islands, with cystic degeneration.

Repeat bronchoscopy and biopsy were performed; the pathology report was as follows: *Gross:* specimen consisted of a small, rounded, dark, soft piece of tissue measuring 7 mm. in diameter. *Microscopic:* sections showed hemorrhagic surface beneath which were irregular epithelial cellular accumulations possessing degenerated centers. *Diagnosis:* The picture here was suggestive of adenoma of the bronchus. Since these tumors are the type which resembled so-called mixed tumors, it is possible that a basal element predominated in certain areas. The prognosis should be the same in either case. On Feb. 23, 1943, a left pneumonectomy was performed through the bed of the fourth rib anteriorly. The vessels and bronchus were ligated individually. As the papillomatous growth was cut through when the bronchus was first divided, another section was removed, thus taking practically all of the left main bronchus. The pathology report was as follows: *Gross:* specimen consisted of the left lung and a portion of the stem bronchus. Two large bronchi could be seen branching from the main bronchus, and one of the two bronchi was thickened about its margin. Serial sections were taken through this entire thickened portion to try to demonstrate the origin of the primary tumor which has previously had a biopsy taken from it. Several enlarged lymph nodes were attached to the separate portion of bronchus and also about the hilum of the lung. Upon dissection of all the large bronchi and many of the smaller bronchi, they were found to be rather markedly dilated and filled with a thick mucoid yellowish purulent-appearing material. *Microscopic:* Sections through the one separate bronchus which was thickened showed a tumor which was of the same type previously described in the biopsies. Portions of the external surface of the tumor were covered by bronchial mucosa. The tumor also extended deeper into the bronchus and in some instances could be seen between the cartilage rings of the bronchus. The tumor was composed of small cells with small dark nuclei, and the cells were arranged about a central cystic area. Sections through the various lymph nodes showed a chronic lymphadenitis. *Diagnosis:* Adenoma of the bronchus, basal-cell type, and chronic lymphadenitis. This tumor showed no evidence of malignancy. The postoperative course was complicated by a low grade persistent fever, but this finally cleared and the patient was discharged twenty-nine days after operation.

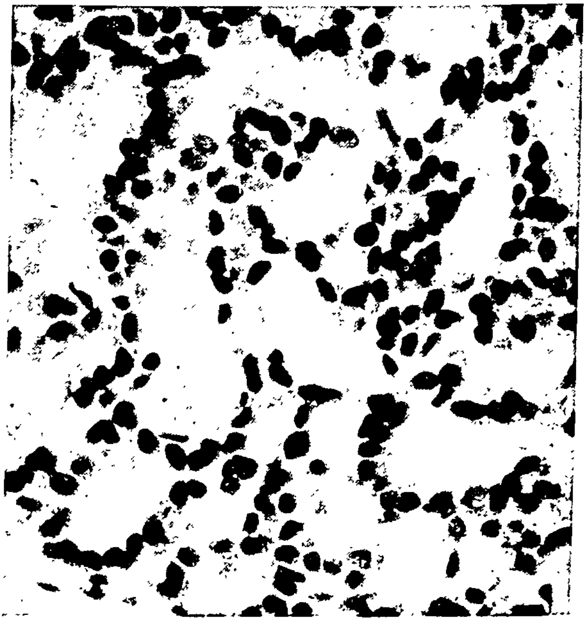

Fig. 2.—Photomicrograph (high power ×435) of a part of Fig. 1, showing cellular structure.

On Feb. 15, 1951, eight years after the pneumonectomy, the patient was readmitted to the hospital, this time complaining of hoarseness, which had been present intermittently for six to eight months. His health had otherwise been excellent; he had been able to work (watchmaker) without difficulty. Examination was negative except for slight hoarseness of the voice, and dullness over the left hemithorax. A roentgenogram of the chest showed a homogeneous density throughout the left hemithorax, with obliteration of the cardiac shadow. The right lung was clear. Laryngoscopy showed the left vocal cord to be immobile. Bronchoscopy revealed a small granular growth in the stump of the left main bronchus. A biopsy was reported as follows: *Gross:* Specimen consisted of a 2 mm. light brown irregular soft fragment of tissue. *Microscopic:* Sections through this tissue showed it to be composed of a loose connective tissue throughout which there were numerous basophilic cells that were fairly uniform in size, shape, and staining characteristics. These cells formed glandular structures which at times were incomplete. In all instances these cells appeared to be well differentiated. *Diag-*

*nosis:* Bronchial adenoma, recurrent. It was believed that he should have left thoracotomy with removal of the recurrent adenoma, which evidently involved the left recurrent laryngeal nerve. Under endotracheal anesthesia the fifth, sixth, and seventh ribs were removed, with the underlying parietal pleura, which was 4 cm. thick, extremely dense, and contained small deposits of calcium. The aorta, bronchial stump, and a portion of the pericardium were bound together in a dense mass. Attempts to separate the structures resulted in severe hemorrhage. It finally became necessary to abandon hope for removal of the bronchial stump. Bleeding was controlled by Gelfoam and hot packs. The chest wall was closed in layers without drainage. Four thousand cubic centimeters of whole blood were administered during the procedure, but nevertheless the patient went into profound shock soon after recovery from the anesthesia. Fifteen hundred cubic centimeters of whole blood given rapidly through an intravenous cannula alleviated the condition. The postoperative course was complicated by severe pain, and a sense of dullness, just below the incision. This was controlled by multiple intercostal nerve procaine blocks. He was discharged on the thirteenth postoperative day to be followed in the Out Patient Department. There had not been any change in his voice. It is anticipated that repeated bronchoscopic removals of the growth will be required.

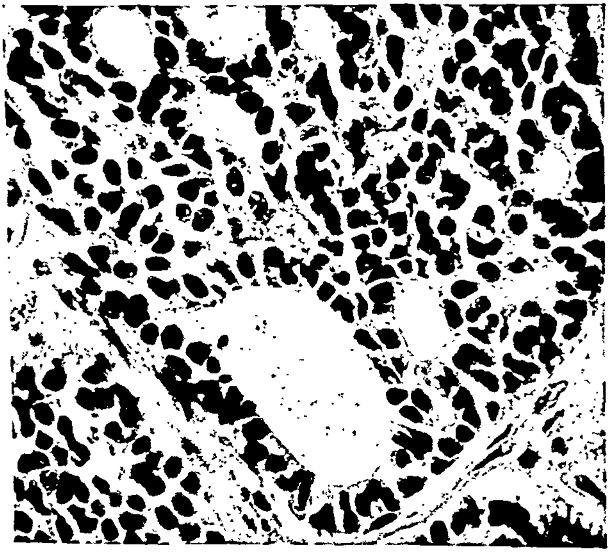

Fig. 3.—Photomicrograph of specimen removed eight years after specimen shown in Figs. 1 and 2. Note similarity of cells (X435).

The recurrence of the tumor was not anticipated. At the original operation the lymph nodes removed with the lung showed no involvement by the tumor. Also, serial sectioning of the bronchus revealed that the site of the second resection was well above the bounds of the tumor. The fact that a recurrence did occur indicates that either undetected local extension was present, or that the regional lymph nodes were invaded at the time of the original therapy. The former seems more feasible in view of the location of the recurrence.

This tumor was recognized as being of mixed origin, in that it showed at various times basal-cell cancer, myxomatous areas, and the typical bronchial adenoma structure; that it was thought to show "no evidence of malignancy" was a misinterpretation. The long quiescent period (eight years) attests to a slow rate of growth, but does not detract from the malignant nature of the tumor.

## SUMMARY

This is a case report of a patient with a bronchial adenoma that showed the characteristics of a mixed tumor, and subsequently recurred eight years after pneumonectomy. It appears that the appellation "mixed tumor adenoma" is more appropriate than adenoma, as applied to such neoplasms of the bronchus.

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# CHONDROMATOUS HAMARTOMA OF THE LUNG

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## INTRODUCTION

PRIOR to 1940 benign cartilaginous tumors of the lung, usually referred to as "chondromas" or "hamartomas," were considered rare and without clinical significance. Recent studies of surgically removed specimens, especially of round, so-called "coin" lesions, indicate that cartilaginous tumors are not uncommon. In fact, they have to be seriously considered in the differential diagnosis of asymptomatic, circumscribed pulmonary infiltrates discovered on routine chest roentgenography. Experience from a single institution may be cited as in indication of the increased frequency of detection of these tumors. In 1945, McDonald, Harrington, and Clagett[1] referred to three hamartomas resected at the Mayo Clinic. Five years later, Lemon and Good,[2] from the same clinic, described the radiographic features of no less than seventeen surgically removed specimens. Among thirty-three clinically silent solid pulmonary lesions removed surgically, Husfeldt and Carlsen[3] found five hamartomas as compared to only four primary carcinomas.

Recently, one of us (M. R.) removed three cartilaginous tumors of the type described as "chondromatous hamartoma." Even in this small group of cases certain features of the tumor were so striking that we were prompted to study the problem further. In addition to the three surgically removed tumors, twenty-eight similar growths found on post-mortem examination at Montefiore Hospital were studied, making a total of 31 cases.

## CASE REPORTS

CASE 1.—S. K., a 54-year-old white man, developed pain in the right shoulder. In the course of a roentgen examination of the chest, a nodule was discovered in the right lung. A routine roentgenogram of the chest taken two years previously had been reported as "negative." Incidentally, the pain was found to be due to subdeltoid bursitis. There were no symptoms referable to the respiratory system. The chest roentgenogram revealed a well-defined, round density measuring about 1 cm. in diameter situated in the apical segment of the right lower lobe (Fig. 1*A*). The nodule was clearly defined on right lateral tomography (Fig. 1*B*).

The patient was admitted to Montefiore Hospital in June, 1948. Bronchoscopy was done on two occasions, but no endobronchial disease found. Smears and bronchial washings failed to reveal tumor cells. Because a definite diagnosis could not be established, exploratory thoracotomy was performed. A firm, round, nodular, yellow mass was found projecting from the lower lobe into the interlobar fissure. It was enucleated without difficulty. The patient made an uneventful recovery, and has remained well for three years.

The specimen consisted of a roughly rounded, sharply circumscribed, hard, grayish-white nodule, 1.5 cm. in diameter, with a whorled, coarsely lobulated surface. Microscopic examination revealed it to be composed predominantly of hyaline cartilage, arranged in a

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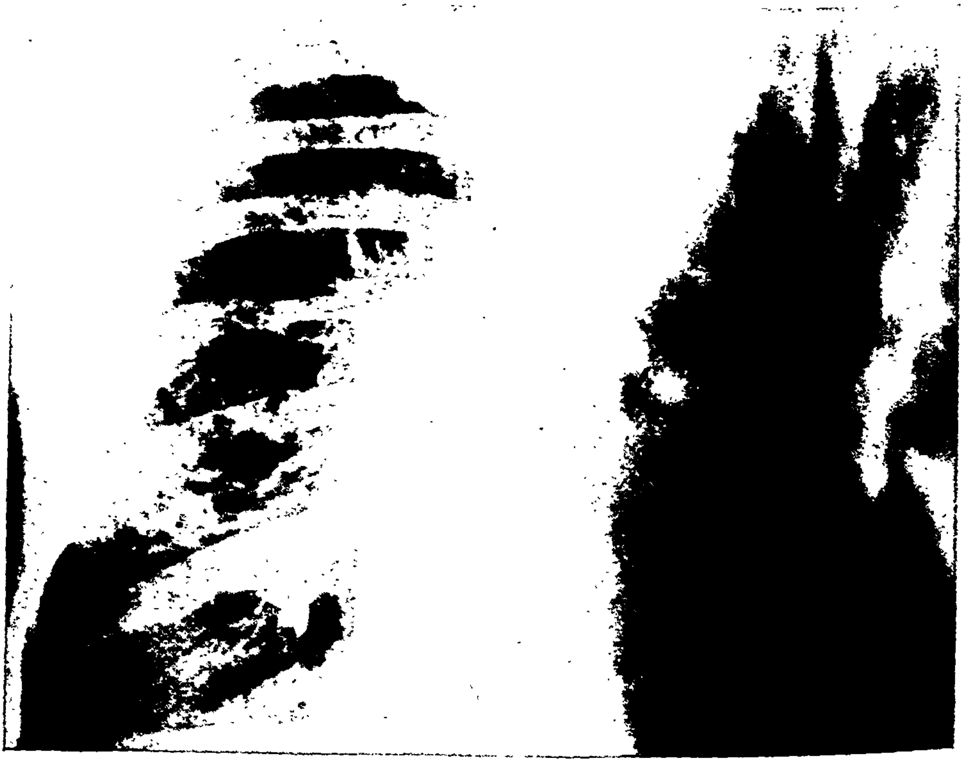
disorderly multilobulated pattern. The areas of cartilage were embedded in loose myxomatous connective tissue which filled the interstices and formed a thin peripheral rim completely surrounding the connected lobules of cartilage. Small bundles of smooth muscle, islands of adult fat tissue, and isolated glandular spaces were encountered in the stroma. The glands were lined by a single layer of epithelium which was cuboidal or columnar, and sometimes ciliated. In places this epithelium was continuous with similar cells, forming an incomplete layer around the surface of the specimen. Small papillae projected into some of the glandular spaces and from some points on the surface. No recognizable lung tissue was found.



A. B.

Fig. 1A and B.—(Case 1.) A, Preoperative chest roentgenogram of a 54-year-old man, showing a small, round mass in the right lung. B, Right lateral tomography. The tumor is located in the apex of the lower lobe.

*Comment:* The radiographic appearance of a circumscribed lesion in a previously normal lung suggested the diagnosis of a primary malignant tumor. The gross appearance of the lesion at operation indicated a benign tumor which could be removed by simple enucleation with preservation of the lobe. Very likely, the mass had been obscured by osseous structures in the x-ray film taken two years previously.

CASE 2.—F. P., a 52-year-old white woman, began to complain of shortness of breath in January, 1949, shortly after a round pulmonary mass was discovered on roentgen examination of the chest. It was soon apparent that the dyspnea was related to a nervous disposition rather than to the pulmonary disease. She was admitted to Lebanon Hospital in May, 1950. Bronchoscopic studies were negative. Radiographic examination of the chest revealed a well-circumscribed, rounded mass, 4 cm. in diameter, in the left lower lobe. In the left lateral projection the mass appeared to be located in the interlobar fissure (Fig. 2). In view of the presence of calcification in the mass, a benign tumor of the lung was suspected. This assumption appeared substantiated by the fact that there had been no change in the size of the mass since its discovery fifteen months previously. At operation, a hard, yellow, nodular mass, 4 cm. in diameter, was found on the interlobar surface of the left lower lobe (Figs. 3 and 4). It protruded above the surface and was easily enucleated. The patient made an uneventful recovery.

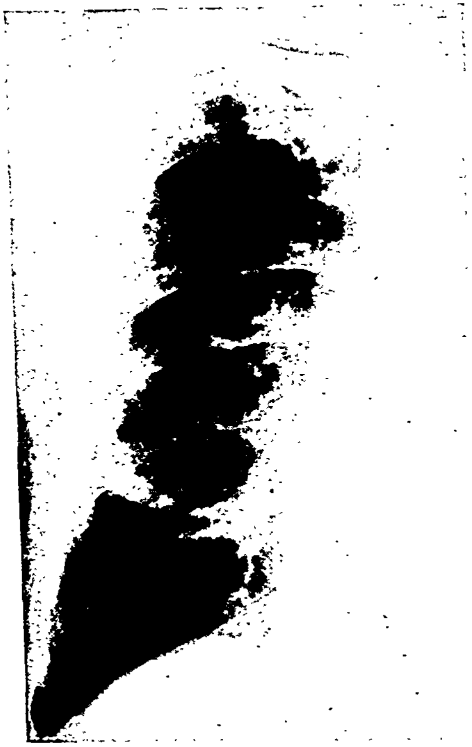
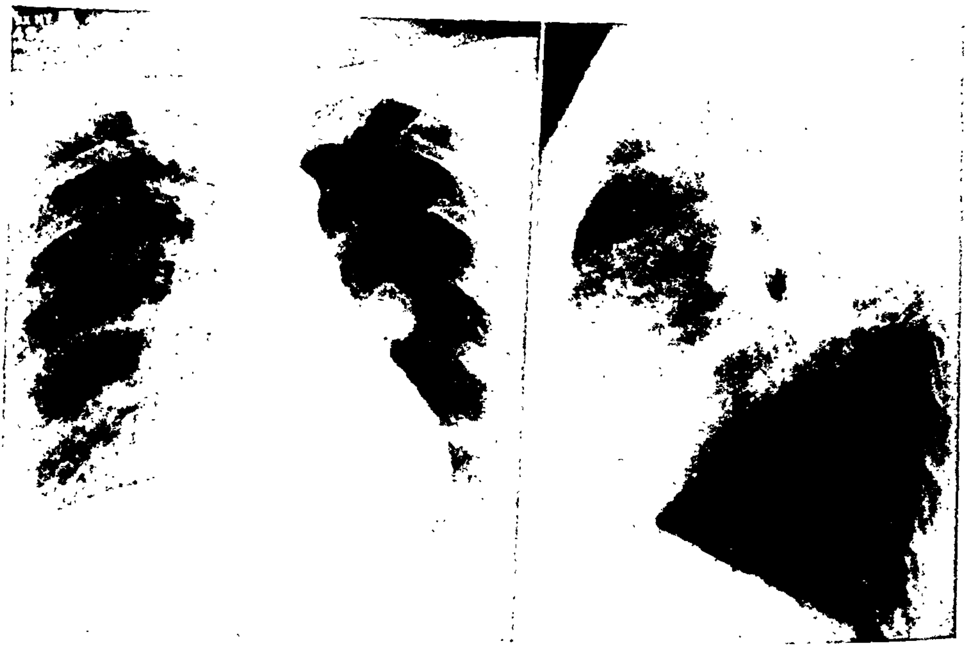


Fig. 1C.—Postoperative roentgenogram following excision of subpleural chondromatous hamartoma.

Microscopic examination of the mass revealed it to be nodular, of rubbery consistency and covered by a thin layer of pleura. Microscopically, it consisted of connected multiform islands of hyaline cartilage which were enmeshed in a mixture of loose myxomatous and dense collagenous fibrous tissue and fat. Bundles of smooth muscle and a few glandular spaces lined by cuboidal and low columnar epithelium were found at the margins and in the fibrous tissue between nodules of cartilage.

*Comment:* While the history and radiographic findings suggested a benign pulmonary neoplasm, a malignant lesion could not be excluded. The tumor was situated beneath the visceral pleura. The gross and microscopic appearance of this mass was similar to that in Case 1. The preoperative impression that the shortness of breath was not related to the lesion has been borne out by the fact that the patient still maintains she is short of breath.



A.

B.

Fig. 2A and B.—(Case 2.) A 52-year-old white woman, complaining of dyspnea. A, Preoperative chest roentgenogram showing a circumscribed tumor in the left lung. B, Left lateral view. Mass is seen in the region of the oblique fissure.

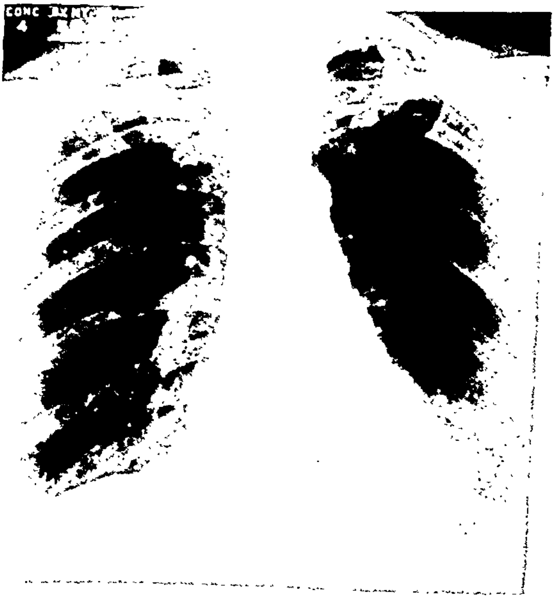


Fig. 2C.—Roentgenogram following operation. A hamartoma was found on the interlobar lobar aspect of the lower lobe.

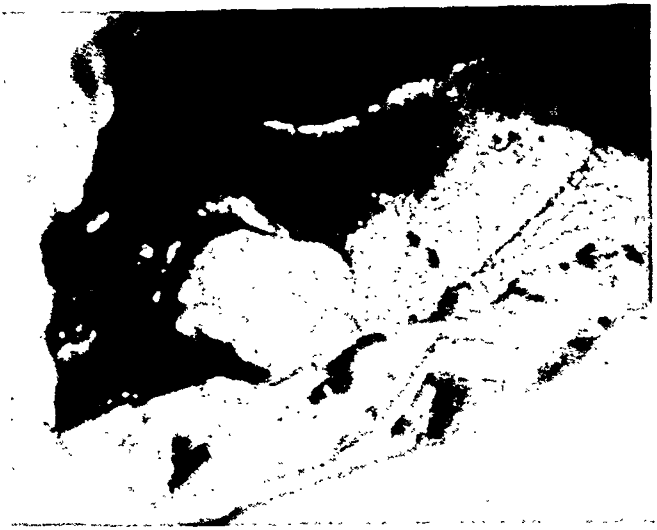


Fig. 3.—(Case 2.) Photograph at operation. A hard, nodular mass was found projecting from the interlobar aspect of the left lower lobe.

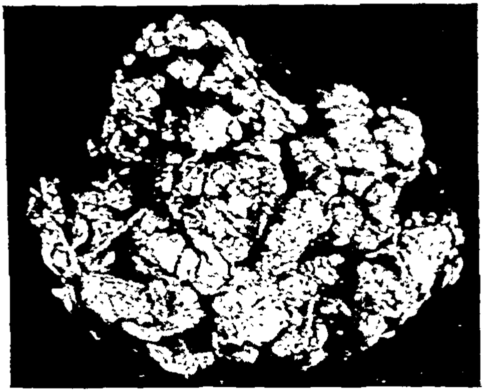


Fig. 4A.

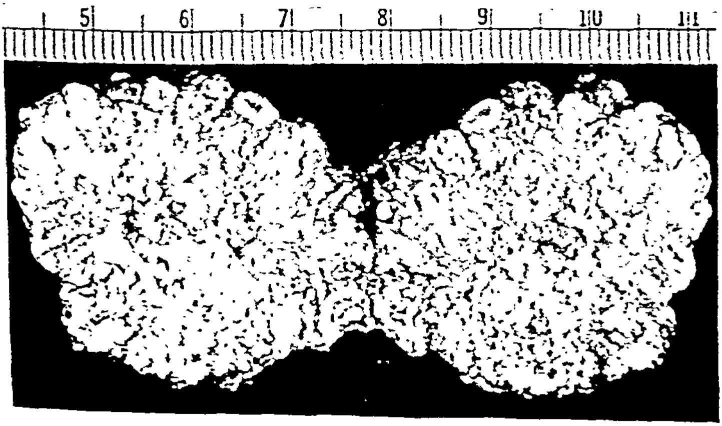


Fig. 4B.

Figs. 4A and B.—(Case 2.) A, Excised hamartoma from the left lower lobe. B, On cross section the mass is composed of numerous lobules with the consistency of cartilage.

CASE 3.—G. D., a 62-year-old, obese, white man, gave a history of bloodspitting of one week's duration. His past and family histories were negative for respiratory disease. He was admitted to the Thoracic Surgical Service of the Morrisania City Hospital in January, 1951.

Roentgenography of the chest revealed a rounded mass in the right lung, 4 cm. in diameter, just above the diaphragm. It was sharply circumscribed and mottled in appearance. Right lateral projection localized the mass to the middle lobe (Fig. 5). Bronchoscopy was negative. A preoperative diagnosis of hamartoma of the lung was made on the basis of the unique radiographic appearance of the mass, as well as on previous experience with such lesions.

At operation, a soft, circumscribed mass about the size of a plum could be palpated in the right middle lobe. It felt like a bean bag. Because of the history of hemoptysis, a right middle lobe lobectomy was performed. The patient made an uneventful recovery.

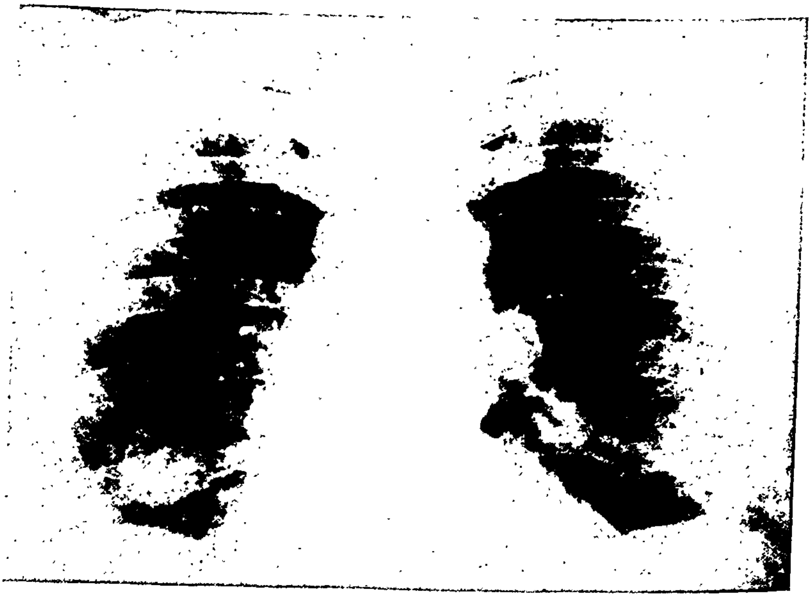


Fig. 5A.—(Case 3.) A 62-year-old white man with a history of hemoptysis. A, Anteroposterior roentgenogram of chest with a circumscribed, nodular mass above the diaphragm.

The specimen was a lobe of lung 13 by 7 by 4 cm. in size (Fig. 6). Its pleural surface was free of visible abnormalities. A compressible, sharply circumscribed, ovoid mass was felt beneath the pleura and measured 4 cm. in diameter. On section, it was separated from the surface by a thin rim of compressed lung tissue which was about 2 mm. in width at its narrowest point. The mass was coarsely lobulated, rubbery to soft in consistency, gray and translucent for the most part, but speckled with denser opaque gray-white foci. It was sharply circumscribed but not encapsulated, and was easily shelled out of its bed, leaving a smooth, deep depression traversed by compressed blood vessels and bronchioles. Relationship to a bronchus could not be demonstrated.

Microscopically, the sharp circumscription without encapsulation was confirmed (Fig. 7). The mass was composed of connected lobules of loose myxomaotus, cellular, connective tissue, the free surfaces of which were covered by a continuous lining, varying from cuboidal to columnar and, in places, ciliated epithelium. In some areas a few mucous glands, adult adipose tissue, and small collections of lymphocytes were noted just beneath the surface epithelium. Small islands of poorly-formed hyaline cartilage were present centrally in only a minority of the lobules. None of the lobules revealed calcification or ossification.

*Comment:* The radiographic appearance of the tumor in this instance was more characteristic of a cartilaginous hamartoma of the lung than in the preceding cases. The mass found within the middle lobe was much softer in

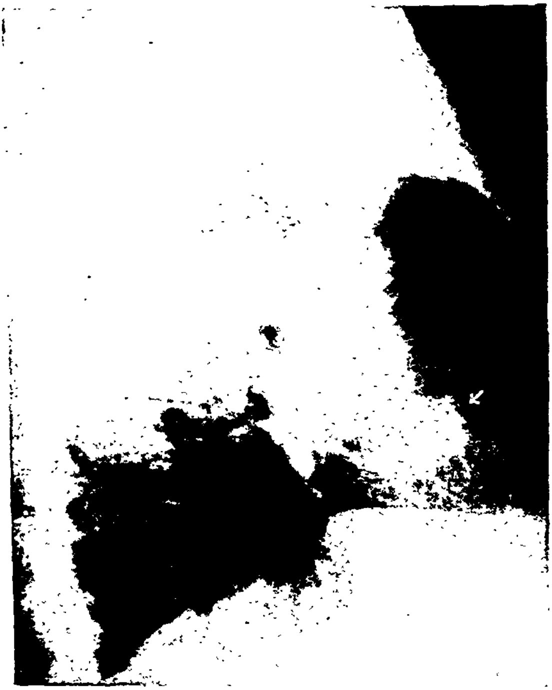


Fig. 5*B*.

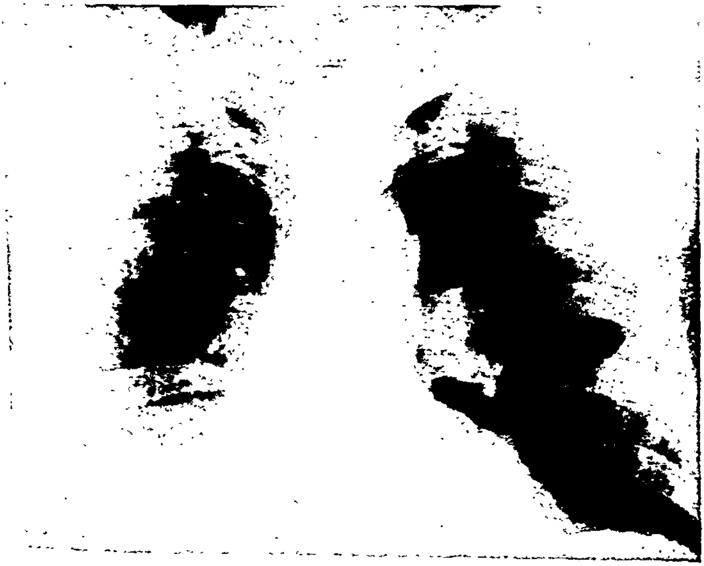


Fig. 5*C*.

Figs. 5*B* and *C*.—(Case 3.) *B*, Right lateral view. The tumor is located in the middle lobe. *C*, Postoperative roentgenogram following right middle lobe lobectomy.

consistency than the lesions in the previous two cases, due to the fact that it contained more myxomatous tissue and less hyaline cartilage. On dissecting the specimen, it was noted that the mass could be completely enucleated from the lobe. Hemoptysis has not recurred despite failure to demonstrate its source in the resected lobe.

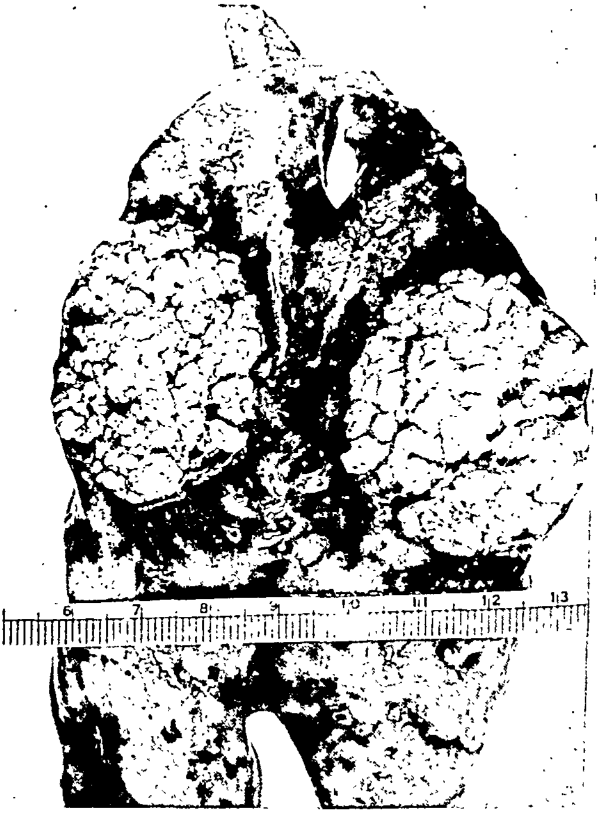


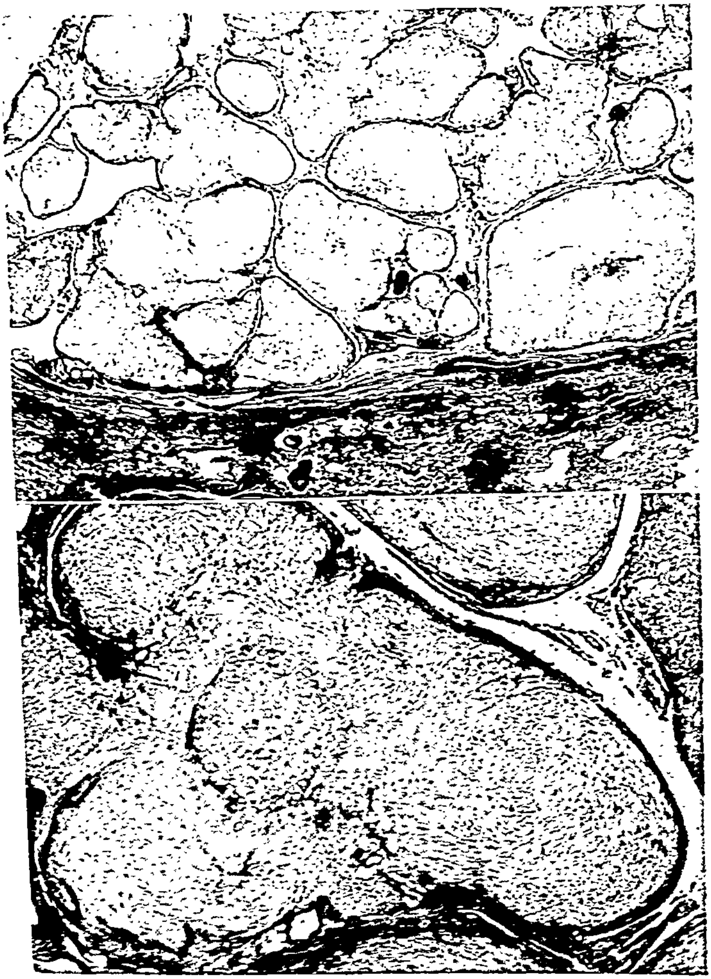
Fig. 6.—(Case 3.) Cross section of excised right middle lobe. Circumscribed tumor is situated below pleural surface consisting of numerous lobules.

## ANALYSIS OF NECROPSY CASES

Solitary cartilage-containing tumors of the lung were found in twenty-eight of 8,800 post-mortem examinations performed at Montefiore Hospital between the years 1914 and 1949. This is an incidence of approximately one in 300 post-mortems. In each instance the tumor was an incidental finding, unsuspected during life in patients who died of various diseases. There were twenty-one males, and seven females (Table I). A comparable sex ratio has been noted by others.

A review of the clinical records of the twenty-eight cases failed to reveal a single instance in which symptoms could be attributed to the cartilaginous tumors. Twelve of the twenty-eight patients had respiratory complaints related to either a primary or secondary pulmonary disease, including tuberculosis, primary or metastatic neoplasms, or cardiac diseases associated with congestive failure. Even if symptoms due to the hamartomas had been present, they would have been very likely overshadowed by those due to the major illness. Roentgenograms of the chest were available in nine of these patients. The involvement of the lungs by other disease processes made it impossible to delineate the cartilaginous tumors with any degree of certainty, even when viewed in the light of the knowledge of the necropsy findings. Of the sixteen patients without primary or secondary involvement of the lungs, roentgenograms of the chest were available in seven. Two revealed shadows which had been over-

A.



B.

Fig. 7.—(Case 3.) *A*, Low-power photomicrograph of hamartoma in the right middle lobe. The lobulated tumor is surrounded by compressed lung (×15). *B*, High-power view showing composition of lobule of myxomatous connective tissue with central area of hyaline cartilage. The surface is covered by a continuous lining of cuboidal or columnar epithelium (×56).

TABLE I. ANALYSIS OF TWENTY-EIGHT CHONDROMATOUS HAMARTOMAS FOUND AT AUTOPSY

| CASE NO. | CHART | AGE | RACE | SEX | CAUSE OF DEATH | X-RAY FINDINGS | LOBE | SIZE | LOCATION IN LOBE |
|---|---|---|---|---|---|---|---|---|---|
| 1 | D. S.<br>8671 | 45 | W | M | Pulm. tbc. | None available | R.U.L. | 3 × 1.5 cm. | Midportion of lobe |
| 2 | B. L.<br>12536 | 61 | W | F | Spinal cord tumor | Round lesion R.U.L. | R.U.L. | 1 × 1 cm. | Subpleural |
| 3 | I. E.<br>32747 | 46 | W | M | Chronic lymphatic leukemia | Negative | L.L.L. | 5 mm. | Subpleural |
| 4 | N. M.<br>34953 | 80 | W | M | Adenocarcinoma of stomach | No masses of pleural fluid | L.L.L. | 1 × 1 cm. | Midportion of lobe |
| 5 | L. C.<br>15898 | 82 | W | F | Heart failure | None available | L.L.L. | ? | Subpleural |
| 6 | H. K.<br>16376 | 65 | W | M | Carcinoma of lung | Multiple nodules | L.L.L. | ? | Wall of bronchiole |
| 7 | W. K.<br>33704 | 71 | W | M | Carcinoma of liver | Mass size of walnut | R.L.L. | 2 × 2 cm. | Attached to medium sized bronchus |
| 8 | E. R.<br>35291 | 67 | W | M | Heart failure | "Density in L.L.L." | L.L.L. | 2 × 2 cm. | Subpleural |
| 9 | S. T.<br>27332 | 66 | W | M | Adenocarcinoma of stomach | None available | L.L.L. | ? | Subpleural |
| 10 | A. G.<br>25215R | 47 | W | M | Acromegaly | Negative | L.U.L. | ? | 1 cm. below pleura |
| 11 | M. N.<br>33523R | 67 | W | M | Adenocarcinoma of rectum | None available | L.U.L. | 1.75 × 2 cm. | Midportion of lingula |
| 12 | M. M.<br>35364 | 72 | W | M | Pulm. tbc. | Plum. tbc. | R.U.L. | 2 × 2 cm. | Subpleural |
| 13 | I. N.<br>32633 | 58 | W | M | Plasma cell myeloma | Multiple nodules | L.U.L. | Microscopic | Midportion of lobe |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| 14 | B. R.<br>39451 | 68 | W | M | Carcinoma of bladder | None available | R.M.L. | 1.5 × 1 cm. | Endobronchial |
| 15 | D. M.<br>43551 | 78 | W | M | Arteriosclerosis | None available | L.L.L. | 5 × 10 mm. | Subpleural |
| 16 | H. B.<br>44248R | 63 | W | M | Carcinoma of esophagus | Diffuse pleural involvement | L.U.L. | 1 × 1 cm. | Midportion of lobe |
| 17 | E. S.<br>39962 | 56 | W | M | Diabetes mellitus, arteriosclerosis | Negative | R.L.L. | 4 mm. | Wall of small bronchus |
| 18 | E. S.<br>41614 | 54 | W | F | Adenocarcinoma of uterus | Negative | R.L.L. | ? | Subpleural |
| 19 | P. K.<br>41606 | 40 | W | F | Adenocarcinoma of breast | Negative | R.U.L. | 1 × 1.5 cm. | Subpleural |
| 20 | F. M.<br>39481 | 63 | W | F | Carcinoma of tongue | Multiple metastases | L.L.L. | 3 × 4 mm. | Midportion of lobe |
| 21 | J. B.<br>36228 | 62 | W | M | Reticuloendothelial sarcoma | Numerous infiltrations | R.L.L. | 8 mm. | Subpleural (diaphragmatic surface) |
| 22 | S. H.<br>37238 | 68 | W | F | Carcinoma of breast | None available | R.U.L. | 2 × 2 cm. | Subpleural |
| 23 | M. J.<br>34109 | 72 | W | M | Adenocarcinoma of stomach | None available | R.L.L. | 1.5 × 1.5 cm. | Midportion of lobe |
| 24 | G. S.<br>25344 | 55 | W | F | Carcinoma of breast | Multiple metastases | R.U.L. | Microscopic | Parenchyma near pleura |
| 25 | J. H.<br>51667 | 63 | W | M | Myelofibrosis, cerebral vasc. accident | None available | R.L.L. | Microscopic | Subpleural |
| 26 | S. L.<br>28163 | 53 | W | M | Rheumatic heart dis. | None available | R.U.L. | ? | Subpleural |
| 27 | J. O.<br>30727 | 39 | W | M | Carcinoma of rectum | Calcified tubercles | L.L.L. | ? | Wall of bronchiole |
| 28 | C. P.<br>47468 | 54 | W | M | Carcinoma of prostate | None available | R.M.L. | ? | Midportion of lobe |

looked in the ante-mortem reading of the films. These corresponded in size and location to the tumors found at necropsy. The failure to demonstrate the tumors in the remaining cases can be ascribed to their small size, or obscuration or both.

TABLE II. ANATOMIC DISTRIBUTION OF 31 CHONDROMATOUS HAMARTOMAS OF THE LUNG

| *Lobar Distribution.—* | | | |
|---|---|---|---|
| *Right Lung:* | | *Left Lung:* | |
| Upper lobe | 7 | Upper lobe | 3 |
| Middle lobe | 3 | Lingula | 1 |
| Lower lobe | 6 | Lower lobe | 11 |
| Total | 16 | Total | 15 |

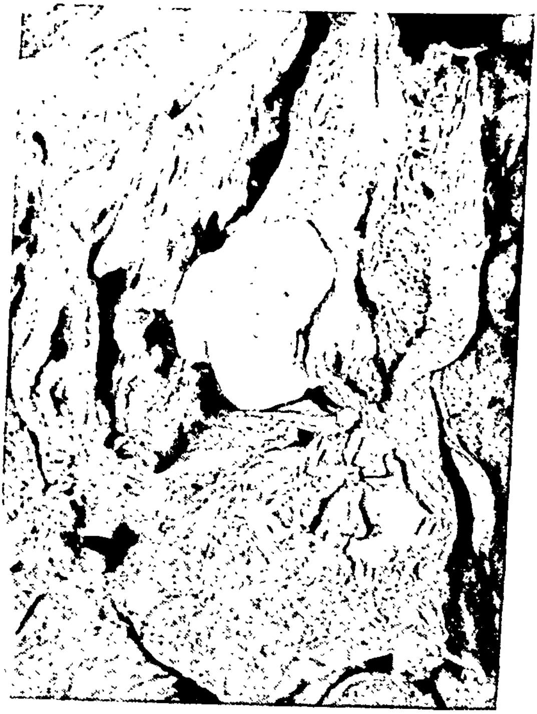


Fig. 8.—Post-mortem specimen (Table I, Case 14) of an endobronchial hamartoma in a branch of the right middle lobe bronchus causing localized saccular dilatation of the bronchus and partial atelectasis of corresponding pulmonary segment.

The anatomic distribution of the growths in thirty-one cases is summarized in Table II. It is apparent that the majority were in the lower lobes and were about equally divided in the two lungs. Twenty-four were located peripherally. Of these, sixteen were immediately subpleural, usually projecting above the surface of the lung. Ten were somewhat deeper, being separated from the surface by a zone of compressed pulmonary parenchyma which varied in width from a few millimeters to a few centimeters. A relationship to a bronchus or large bronchiole, which could be interpreted as a true connection, was found in five cases. In four of these the tumor merged imperceptibly with the wall of a large bronchiole which, however, was neither compressed nor distorted. In one case (Fig. 8) the mass formed an obstructing intraluminary polyp in a major branch of the middle lobe bronchus. It was 1.5 cm. in diameter and caused a localized saccular dilatation of the bronchus, with partial atelectasis of the corresponding pulmonary segment.

The majority of the tumors were small, varying from 1 to 2 cm. in greatest diameter. The smallest was 0.4 by 0.3 cm., the largest 3.0 by 1.5 cm. Their macroscopic appearance recorded by as many prosectors as there were tumors was monotonously alike, and furnished striking evidence that these tumors are of a similar nature. On palpation, they were rounded or ovoid, sharply circumscribed, smooth, or coarsely lobulated. While most of the tumors felt hard and unyielding, some were rubbery and compressible. The cut surface resembled cartilage, the uniformity of which in some cases was interrupted by gritty, calcified, or ossified spots and streaks. With few exceptions, the masses were free of attachments to the adjacent parenchyma and could be easily enucleated, leaving a smooth defect. In some cases the macroscopic appearance was sufficiently characteristic to allow gross differentiation of the tumor from coexistent nodular cancerous metastases or tuberculous lesions elsewhere in the lung.

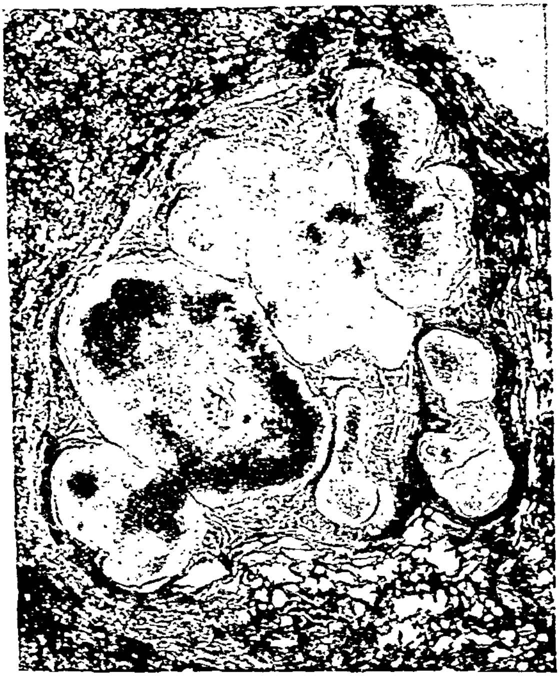


Fig. 9.—Low-power photomicrograph (×65) of a small hamartoma showing multiple, closely crowded, connected lobules, the most prominent component of which is a solid central island of cartilage. The tumor is not encapsulated but surrounded by compressed lung tissue (×15).

Microscopic preparations of each of the tumors were available and reexamined for the purpose of this study. Their histologic appearance was unique.

They were composed of mesodermal and epithelial elements in various proportions, but in a well-defined and uniform architectural pattern. They consisted of multiple, closely crowded, connected lobules, the most prominent component of which was a solid central island of cartilage (Fig. 9). In the majority, foci of calcification were present in the cartilage; less frequently isolated spicules of bone were encountered. The cartilaginous core was surrounded by a zone of fibrous connective tissue of variable width and structure. Usually, it was fibrillar and myxomatous in appearance but, at times, compact and collagenous.

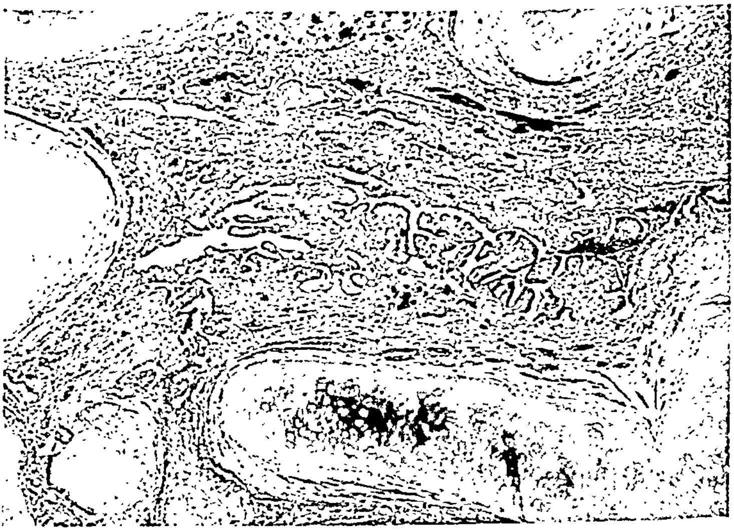


Fig. 10.—High-power photomicrograph showing clefts between lobules with glandular spaces arranged in an irregular, papillary fashion and lined with cuboidal epithelium (×56).

A condensation of cellular elements in the transition zone, simulating perichondrium, was found in some cases. The free surfaces of individual lobules were covered by a layer of epithelium which varied from a low cuboidal type to tall and columnar, with or without cilia. This epithelium was indistinguishable from that lining normal subdivisions of the bronchial tree, and usually dipped into the underlying stroma to form distorted glands. Not infrequently, it protruded into the clefts between lobules, or formed glandular spaces in an irregular, blunt, papillary fashion (Fig. 10). Small sheets of adult adipose tissue were present just beneath the surface in the majority of cases. Strands of smooth muscle bundles and isolated lymphoid follicles were encountered less frequently. A definite capsule was not present. Adjacent alveolar septa, bronchioles, and small blood vessels were stretched and compressed. Histologic stigmas of malignancy were absent in all cases.

## DISCUSSION

The vast majority of hamartomas of the lungs are found accidentally at post-mortem examination. In recent years, with the wider use of routine radiographic examinations of the chest and the attitude that asymptomatic solitary lesions of the lung, which otherwise defy diagnosis, warrant exploratory thoracotomy, increasing numbers are being removed surgically. It is of interest that two of the three patients in whom this lesion was found at operation presented symptoms referable to the respiratory system. Yet, in both cases it is difficult to attribute the symptoms to the tumor. The difficulty in breathing in Case 2 has persisted following the removal of the mass. The middle lobe removed in Case 3 failed to reveal any source for bleeding which, however, has not recurred since the operation. It is very likely that the symptoms were not caused by the tumor in either instance.

Symptoms which can be attributed to hamartomas are absent, unless they produce bronchial obstruction or are large enough to compress adajacent structures. This feature can be correlated with the size and location of the majority of the tumors, both in our material and in the cases reported by others. Thus, we found a true connection with a bronchus or large bronchiole in only five cases. In four of these there was no compression. In one case the growth formed an obstructive endobronchial polyp (Fig. 8). Location within a bronchus is uncommon, this representing the fourteenth case of this type which has been reported.

Although on occasion the identity of the lesion may be suspected from the roentgen findings, only exploratory thoracotomy and pathologic examination of the tumor can substantiate the diagnosis. The radiographic findings illustrated in Case 3 led to a correct preoperative diagnosis. The roentgenogram accurately reflected the gross structure of the mass. Except in the unusual case in which the growth is located within a bronchus, additional diagnostic methods including bronchoscopy and cytologic study of material aspirated from the bronchus give little, if any, information because of the structure and peripheral location of the lesion. However, such tests are essential in order to rule out carcinoma or other diseases.

Hamartomas are usually ideally suited for surgical extirpation with minimal loss of functional lung tissue. Most hamartomas are peripherally located and easily accessible for enucleation through a small pleural incision. A few are centrally located and may require lobectomy, particularly if malignancy is suspected at operation. A rare form of massive hamartoma, almost replacing a lobe, has been encountered in a newborn infant at autopsy.[7] Objections to simple enucleation on the grounds of potential malignancy are theoretical on the basis of available data. With the possible exception of the case reported by Simon and Ballon,[9] there is no recorded instance of a true malignant neoplasm developing from a cartilaginous hamartoma of the lung. This statement is predicated on the opinion that cartilaginous hamartomas are biologically different from bronchial adenomas. The relationship of the latter to malignant neoplasm is well known. When located superificially, their appearance is sufficiently characteristic to allow recognition at operation. Where doubt exists, examination of frozen sections quickly establishes the diagnosis. No other tumor of the lung presents a similar histologic picture.

This study yields no original data to help elucidate the pathogenesis of the tumor of the lung under discussion; nor is it concerned, except incidentally, with problems of nomenclature and classification. Most of the cases in our necropsy group were originally designated as chondromas. While their most striking component is cartilage, they invariably contain other mesodermal and epithelial elements. We were not able to find a single pure chondroma in the 8,800 necropsies reviewed. This might have been predicted from the accumulated experience of others, without necessarily denying that true chondromas exist in this location. It is significant, however, that on critical examination almost all reported extrabronchial "chondromas" of the lung are identical, consisting of a mixture of mesodermal elements in addition to cartilage. The current consensus of opinion is that these growths are tumorlike malformations resulting from an abnormal mixture of normal components of an organ. Some prefer the designations hamartochondroma or chondromatous hamartoma of the lung to differentiate these tumors from the vascular malformation of the liver to which the term was originally applied by Albrecht[10] and from the group of pulmonary tumors designated as adenomas.

## CONCLUSIONS

1. Three cartilaginous hamartomas of the lung removed surgically, and twenty-eight found at post-mortem examination are reported.
2. Because of the increasing frequency with which this tumor is being encountered, the lesion must be considered in the differential diagnosis of all solitary, rounded pulmonary lesions.
3. A correct diagnosis may occasionally be made from the roentgen appearance of the lesion, but a definitive diagnosis can be made only by exploratory thoracotomy and examination of the mass.
4. Because of their unique microscopic appearance and their usually superficial location in the lung, most cartilaginous hamartomas can be readily identified in the operating room and removed by simple enucleation. Lack of familiarity with the characteristics of this lesion may result in needless sacrifice of lung tissue at operation.

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# PULMONARY RESECTION AND UNDECYLENIC ACID IN SYSTEMIC BLASTOMYCOSIS

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IN 1894 Gilchrist[1] described the clinical picture of blastomycosis and identified the causative organism. He also suggested the use of potassium iodide in its therapy.

Numerous reviews of the literature give a complete clinical and laboratory picture of the disease.[1, 2] Two forms are recognized, the cutaneous and the systemic. The cutaneous type is characterized by single or multiple pyogenic granulomatous skin lesions. In the systemic type, single or multiple granulomatous lesions are present in the internal organs. About 50 per cent of all cases fall into each group.[1] The lungs are involved in over 90 per cent of the systemic group. The pulmonary focus is usually responsible for dissemination.[2] Cutaneous disease rarely results in dissemination.

The mortality rate of systemic blastomycosis is accepted as 90 per cent within three to five years after the onset of the disease.[1] Because methods of treatment have proved unsatisfactory, we have used previously unreported types of treatment in two patients. The first was a patient upon whom a pneumonectomy was done with the preoperative diagnosis of systemic blastomycosis which was localized to the right lung. A postoperative diagnosis was made in the second patient after the left lung had been removed as a palliative procedure with the mistaken diagnosis of carcinoma. Postoperatively he has been treated with undecylenic acid given orally.

Hoffman and associates[3] in 1939 showed the fungistatic effect of many fatty acids and their salts for certain nonpathogenic fungi. Their work resulted in the introduction of sodium and calcium propionate into the baking and dairy industry.

Keeney[4] was stimulated by Hoffman's work, and in 1943 studied the in vitro fungistatic and fungicidal effect of sodium propionate on common pathogens. At a pH of 5.5 complete fungistatic effect was obtained for blastomyces dermatitidis at a drug concentration of 0.0125 per cent. Results were poor at a pH of 7.4. Fungicidal activity was also demonstrated. Promising results were obtained for other pathogens including *Torula histolytica, Coccidioides immitis,* and *Histoplasma capsulatum.* In the same year Keeney and Broyles[5] reported good clinical results with sodium propionate on several varieties of superficial fungus infections.

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[*] Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the results of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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Keeney's[6] next work, reported a year later, had to do with the fungistatic and fungicidal activity in vitro of sodium valerate, sodium caproate, sodium caprylate, sodium caprate, and sodium undecylenate. Hoffman and coworkers[3] had reported that the fatty acids of these salts were most effective for nonpathogenic fungi at pH 7.4. These fatty acid salts were tested in vitro against fungi met in clinical practice as the common causes of the superficial and the deep mycotic infections. They showed that the salts of fatty acids acquire fungicidal activity as their chain is lengthened. The two highest chain salts, sodium caprate and sodium undecylenate, were shown to be the most promising fungicides. They were also the best fungistatic agents against the fungi that produce deep mycotic infections. Each produced maximum fungistatic activity at a concentration of 0.1 per cent (100 mg. per cent).

Thus we had evidence of the value of the fatty acids and their salts. Perlman's[7, 8] work reported in 1949 provided us with definite evidence that large amounts of undecylenic acid could be taken orally. Nineteen of his forty-one patients showed mild gastrointestinal disturbances when up to 19.8 Gm. of undecylenic acid were taken daily for long periods. He used it with controversial results in psoriasis and neurodermatitis. After treatment was started, we obtained further evidence of the drug's low toxicity from the work of Rattner.[9] He reviewed 1,100 cases of psoriasis treated with undecylenic acid. No figures were given as to dosage or form in which the drug was taken, but gastrointestinal upsets were said to have been frequent.

Before treatment was started, we determined the in vitro effect of undecylenic acid on cultures of *Blastomyces dermatitidis* from four patients (Table I). In all cases the organism failed to grow in a liquid broth mixture with a concentration of 10 mg. per cent of the drug.

Keeney and associates[6] had obtained complete inhibition of blastomycosis dermatitidis at 100 mg. per cent. This discrepancy in their and our results may be accounted for by three factors. They used the mold form of the organism in a solid medium medicated with sodium undecylenate. We used the yeastlike form of the organism in a liquid medium medicated with undecylenic acid. A liquid medium and the yeastlike or tissue form of the organism seemed more completely to simulate the conditions found in the systemic form of the disease. We believed that our collected evidence and results warranted trial of the drug.

TABLE I. SENSITIVITY OF BLASTOMYCES DERMATITIDIS TO UNDECYLENIC ACID

| | STRAIN NUMBER | | | |
|---|---|---|---|---|
| | 1 (MG. PER CENT) | 2 (MG. PER CENT) | 3 (MG. PER CENT) | 4 (MG. PER CENT) |
| 100 per cent inhibition | 10 | 10 | 10 | 10 |
| 90 per cent inhibition | | | 9 | |
| 75 per cent inhibition | | | 8 | |
| 50 per cent inhibition | 9 | | 6 | 9 |
| 25 per cent inhibition | | 9 | 4 | |

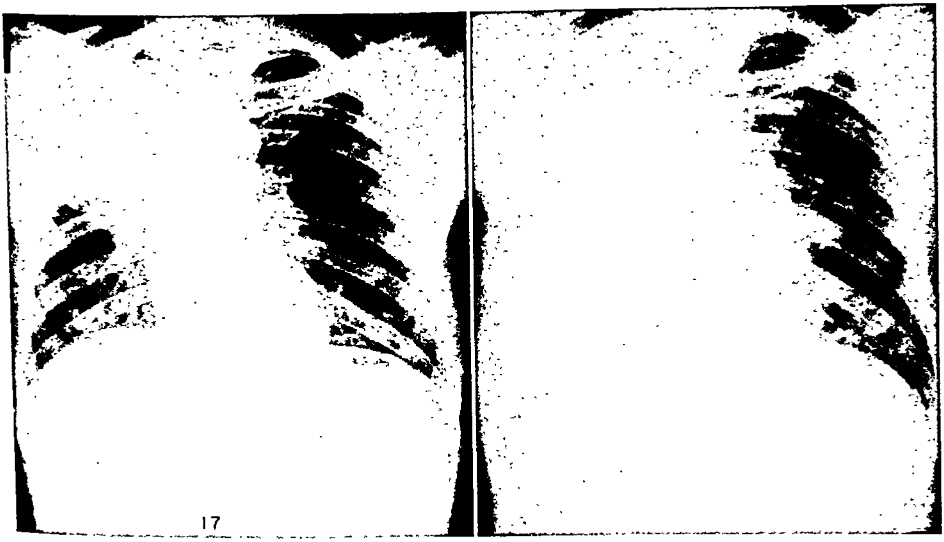
Sensitivity tests with undecylenic acid on four different strains of blastomyces dermatitidis. Tests done in tryptose phosphate broth (Difco pH 7.3) with the addition of 5 per cent horse serum. Incubated at 37° C. for seven days. Organisms tested in the yeastlike or tissue form.

Because of the previously unreported forms of therapy used, we are presenting two cases.

## CASE REPORTS

Case 1.—V. R., a 34-year-old Negro man, was admitted Dec. 17, 1949. Before World War II, he had done steel mill and construction work as a laborer. He served in the European and Southwest Pacific theaters. His past history was negative and previous health good.

The present illness began in 1945. On admission the original right chest pain, hoarseness, and streaking were still present. He also complained of fatigue, weakness, cough, fever, night-sweats, anorexia, and 20 pounds in weight loss. With the exception of short periods of improvement of one or more symptoms, there had been gradual retrogression during this four-year period.



*A.* *B.*

Fig. 1.—(Case 1.) Preoperative (Jan. 5, 1950) and postoperative (April 19, 1950) films. *A*, Dense shadow in right upper lobe and superior segment of lower lobe. Pneumonectomy confirmed preoperative diagnosis of blastomycosis. *B*, Perihilar infiltration extending into lingula and left lower lobe. Autopsy confirmed clinical impression of progression of spread of blastomycosis in left lung.

Physical examination showed a chronically ill patient with temperature range of 100° to 103° F. daily. Examination of the right lung showed slight impairment of resonance, diminished breath sounds and posttussive râles (Fig. 1,*A*). The chest plate showed a pneumonic lesion in the right upper lobe.

Admission blood studies showed a red count of 5,200,000, while the hematocrit was 46 and the hemoglobin 97 per cent. The total leukocyte count was 17,200, with 80 per cent polymorphonuclears, 18 per cent lymphocytes, 1 per cent monocytes, and 1 per cent eosinophils. During the rest of the preoperative course the eosinophil count did not exceed 3 per cent. The sedimentation rate (Wintrobe corrected) was 32 mm. per hour. Urinalysis showed a trace of albumin. The actual vital capacity was 2,600 c.c. compared to a normal for him of 4,550 cubic centimeters.

Smears and cultures were negative for tubercle bacilli, but positive for *Blastomyces dermatitidis*. Both the blastomycosis skin test and complement fixation test were negative. Electrocardiography was negative. Bronchoscopy showed a small amount of purulent material in the right upper lobe orifice.

After nineteen days of study, hospital care, and preparation, a right pneumonectomy was done on Jan. 6, 1950, in the face-down position, using cyclopropane anesthesia.

*Figs. 2A and 2B.*—In the gross specimen the upper lobe and part of the lower lobe were found to be firm in consistency. Cut sections showed pinkish-gray granulomatous areas with yellow foci. Numerous small cavities with firm walls were present.

*Figs. 3A and 3B.*—Microscopic examination disclosed numerous giant cells, epithelioid cells, and polymorphonuclear cells. Areas of pneumonia, pseudo-tubercles and micro-abscesses were interspersed. Numerous double contoured yeastlike organisms, about 20 microns, were identified in the lung.

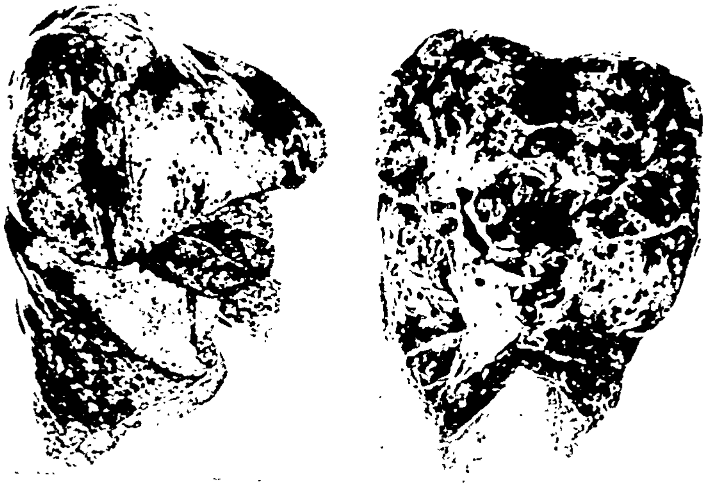


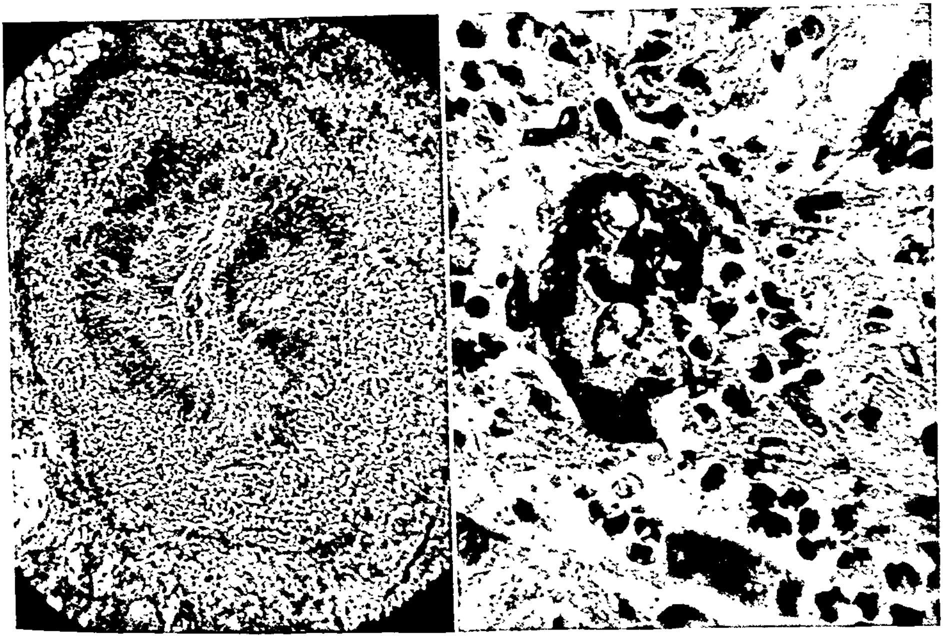
Fig. 2.—(Case 1.) Gross specimen of right lung (Jan. 4, 1951). *A*, Note consolidation of right upper lobe. *B*, Section of lobe shows numerous small cavities and confluent blastomycosis granulomas.

*Figs. 4A, 4B, and 4C.*—Culture on blood agar at 37° C. produced numerous small, heaped, wrinkled, waxy colonies after two weeks' incubation. Microscopic examination of the culture showed budding double contoured yeastlike organisms identical with those seen in the lung. Culture on Sabouraud's medium at room temperature produced a white cottony, aerial growth, typical of blastomyces dermatitidis.

A bronchogenic spread to the left lung was evident ten days after surgery. Since the skin test was negative desensitization with the blastomycosis vaccine was not done before potassium iodide therapy was started on Jan. 11, 1950. Treatment started with 30 drops daily. The patient was taking 300 drops (15 Gm.) by Feb. 28, 1950, when it was necessary to stop the treatment because of nausea. Small doses were again started but were stopped on April 17, 1950, when the dose had reached 420 drops daily. The patient's general condition was at this time much worse. The skin test had become weakly positive, and the complement-fixation test was positive to a 1 to 32 dilution. Desensitization was considered but subsequent skin tests were negative.

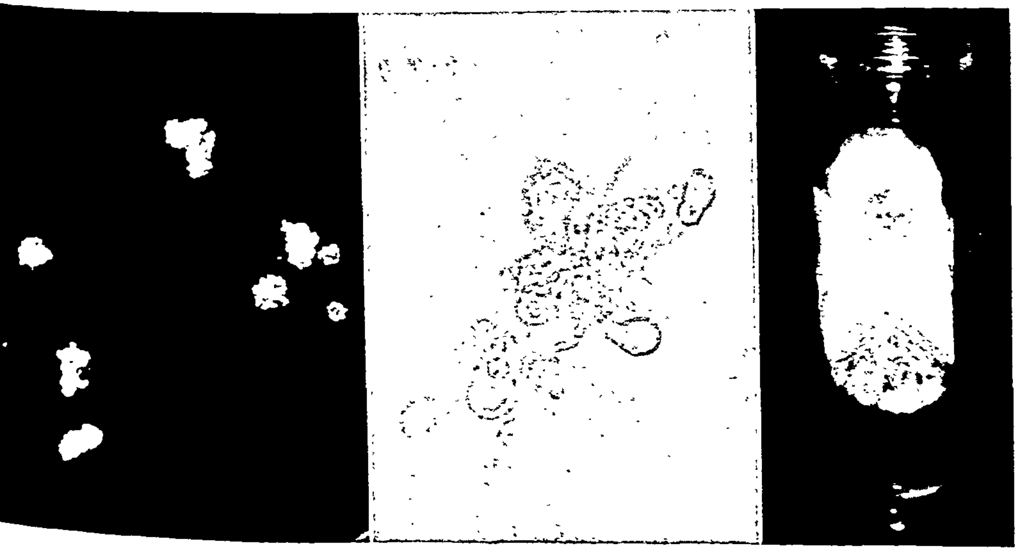
*Fig. 1B.*—Shortly before death, six months after the pneumonectomy, a film of the left lung showed progression of the disease. Exudative disease was seen throughout the central portion of the lung field. The costophrenic angle and the periphery remained comparatively clear.

Autopsy showed blastomyces dermatitidis granulomas in the left lung, the stump of the right bronchus, and empyema in the right pleural cavity. Organisms were also recovered from granulomas in the left kidney and heart.



A. B.

Fig. 3.—(Case 1) Photomicrographs of histologic preparations from right upper lobe. *A*, Low-power examination of blastomycosis tubercle. Tubercle indistinguishable from a tuberculosis process in this low-power magnification. *B*, High-power photograph (detail of Fig. 3*A* demonstrating double-contoured Blastomyces dermatitidis organisms in a giant cell.



A. B. C.

Fig. 4.—(Case 1) Smear and cultures, blastomyces dermatitidis. *A*, Blood agar at 37° C., two weeks. Note numerous small, heaped, wrinkled, waxy, cream-colored colonies. *B*, High-power photomicrograph, fresh potassium hydroxide preparation. Note budding, spherical, double-contoured, yeastlike organisms identical with those seen in the lung. *C*, Culture on Sabouraud's medium at room temperature. Note white cottony, aerial growth, typical of *Blastomyces dermatitidis*.

*Comment.*—This case of systemic North American blastomycosis presented a typical clinical course with characteristic signs, symptoms, and laboratory studies. The man's condition had gradually deteriorated during four years, with the disease apparently localized in the right lung. At the time of surgery the negative skin test and negative complement-fixation test suggested a doubtful prognosis.[10] Both tests became weakly positive after surgery. The development of the positive skin test indicates that the patient was developing some resistance. Iodide therapy did not help his general condition. We believe that if the bronchogenic spread had not occurred, life would have been considerably prolonged as a result of the pneumonectomy.

CASE 2.—A. P., a 40-year-old white man, was admitted Dec. 16, 1950, from the Veterans Administration Hospital, Fayetteville, N. C. He had entered there Oct. 24, 1950. His general health had been good up to the present illness. Two years before World War II service in 1944 he had worked in the Army Quartermaster Department at Goldsboro, N. C. Since the war he has continued as a watchmaker in Goldsboro. Military service was in the United States.

The present illness began at 4:30 A.M., Oct. 13, 1950, when he was awakened with pleuritic pain in the left anterior chest wall. He soon developed a cough productive of a small amount of gray-black sputum. Anorexia, weakness, and weight loss were progressive. Ten days after the onset he was admitted to the Fayetteville Hospital. Penicillin and aureomycin were given with no improvement. The temperature curve continued between 99 to 101° F. Smears and cultures were negative for tubercle bacilli.

On admission to Oteen, as a possible carcinoma of the left lung, all symptoms and signs continued. There had been a 20-pound weight loss since the onset. Examination of the right lung was negative. The left anterior chest wall showed limitation of motion. Dullness and decreased breath sounds were present above the third rib on the left. Blood pressure was 130/90, and pulse was 100.

*Fig. 5A.*—A chest plate of Jan. 3, 1951, showed atelectasis of the anterior segment of the left upper lobe with no evidence of cavitation. There had been no change since admission.

Laboratory findings showed a hypochromic normocytic anemia. The red count was 4,400,000, while the hematocrit was 44, and the hemoglobin 11.6 Gm. The total leukocyte count was 13,800, with 60 per cent polymorphonuclears, 32 per cent lymphocytes, and 8 per cent monocytes. In several other preoperative white counts eosinophils never exceeded 3 per cent. The sedimentation rate was 50 mm. per hour (Wintrobe corrected). Urinalysis showed a trace of albumin. The actual vital capacity was 2,500 c.c. compared with a normal for him of 4,600 cubic centimeters. Nine smears and one culture were negative for tuberculosis. Fungus smears and cultures were not made.

A small amount of pus in the left upper-lobe orifice was present at bronchoscopy. Bronchial washings were obtained and showed no evidence of malignant cells. Cytologic study was made on four sputum specimens. One was suggestive for carcinoma.

After nineteen days of study with eighteen days of streptomycin and six days of penicillin, there was no change in the clinical course. Exploratory thoracotomy was done Jan. 4, 1951, and confirmed the impression of atelectasis of the anterior segment of the left upper lobe. The segment contained a 4 cm. mass and with the lingula was densely bound to the mediastinum and anterior chest wall. The impression was that we were dealing with a carcinoma of the lung. It was necessary to remove a portion of the pericardium. The gross specimen showed atelectasis of the anterior segment with a mass of granulation tissue 4 cm. in diameter extending along and occluding the distal portion of the bronchus.

Microscopic examination showed a chronic granuloma characterized by agglomerations of epithelioid cells, giant cells, polymorphonuclears, and micro-abscesses. Yeastlike organisms were seen. Culture on blood agar and Sabouraud's media produced the typical colonies of *Blastomyces dermatitidis*.

The immediate postoperative course was stormy. Transient patchy atelectasis was present. Auricular fibrillation developed and was controlled with quinidine. There was marked dyspnea and mental confusion. The temperature curve was a septic type for thirty days. General condition was fair with little dyspnea after one month. Streptomycin, penicillin, and chloromycetin had been given for the first three postoperative weeks.

A blastomycosis skin test and complement-fixation test for blastomycosis were both negative two weeks after the resection. Fluid recovered from the left chest cavity Jan. 19, 1951, was positive for *Blastomyces dermatitidis* on culture. At the end of the first postoperative month the white count was 20,200, with 85 per cent neutrophils, 10 per cent lymphocytes, 4 per cent monocytes, and 1 per cent eosinophils.

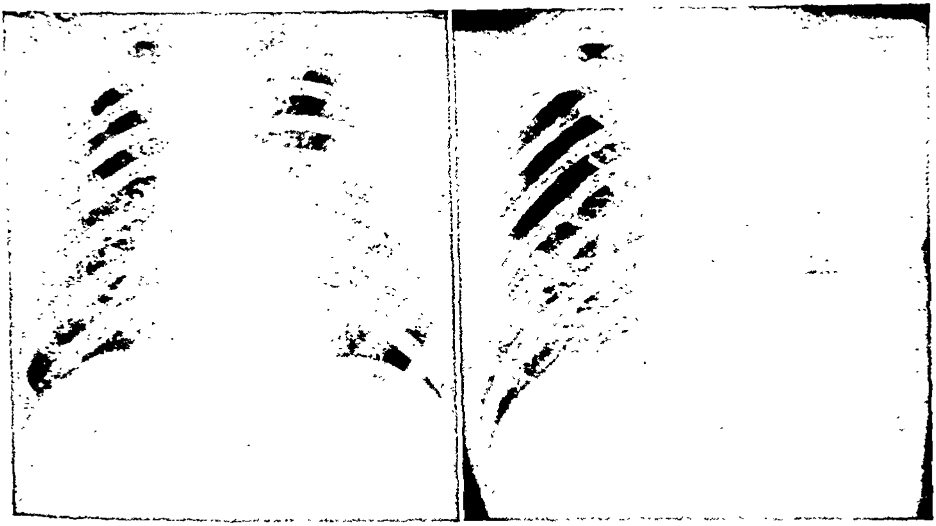


Fig. 5.—(Case 2.) Preoperative (Jan. 3, 1951) and postoperative (July 20, 1951) films. *A*, Dense shadow in anterior segment of left lung. No disease in right lung. Pneumonectomy (Jan. 4, 1951) showed atelectasis. The segment contained a 4 cm. mass of blastomycosis granulomatous tissue. *B*, The right lung is free of disease and the left thoracic cavity shows complete haziness with no evidence of disease.

Three weeks after surgery potassium iodide was started with nine drops daily. On March 7, 1951, when he was taking 54 drops daily, nausea developed and treatment was stopped. Desensitization had not been done since the skin test was negative. An emergency leave was given March 10, 1951, and he returned March 18, 1951, with serum jaundice. Because of the jaundice, potassium iodide was withheld. On April 21, 1951, he developed a papulopustular rash. This involved his face, scalp, gums, neck, ears, and both pectoral regions. The onset was associated with fever up to 101° F., which persisted for four days. Smears of the pustules were positive for *Blastomyces dermatitidis.*

On May 3, 1951, the patient was started on oral undecylenic acid capsules (0.44 Gm.) four, three times daily, for a total of 5.28 Gm. The white count at this time was 13,450, with 73 per cent neutrophils, 20 per cent lymphocytes, 4 per cent monocytes, and 3 per cent eosinophils. By the end of the first month the dose had been increased to 10.5 Gm. daily. The facial pain associated with the rash disappeared within ten days. Night sweats which had persisted soon ceased, appetite improved, and the patient began to gain weight.

After one month of treatment the skin lesions were confined to the forehead and shoulders. They now resembled small, healing furuncles. Smears and cultures were negative. The white count was 18,150 with 49 per cent neutrophils, 33 per cent lymphocytes, 9 per cent monocytes, and 9 per cent eosinophils. The sedimentation rate was 10 mm. per hour. The blood level of undecylenic acid was determined by adding 1 part of the patient's serum to 5 parts of the media mentioned in Table I. This mixture resulted in 100 per cent inhibition to growth of the organism. Since it had been shown that the organism was completely inhibited by 10 mg. per cent of the drug, we now knew that the blood contained approximately 50 mg. per cent of undecylenic acid.

His condition was so good June 14, 1951, that he was given leave with instructions to take undecylenic acid at home. He returned from leave July 20. At this time a few very small pustules were present on the face. No organisms were found on biopsy, smear, or culture. He had no complaints. The weakness had disappeared completely and his appetite was very good. The blastomycosis skin test was now positive but the complement fixation test was *still negative.*

*Fig. 5B.*—A chest plate dated July 20 shows no evidence of disease. He was again instructed to take undecylenic acid at home.

When last seen Aug. 31, 1951, the hemoglobin was 17.3 Gm., the hematocrit 52, and the white count 9,600, with 58 per cent neutrophils, 35 per cent lymphocytes, 7 per cent monocytes, and no eosinophils. The sedimentation rate was 19. The chest plate was negative. A few furuncles which were present over the shoulders were negative to smear and culture for *Blastomyces dermatitidis.* He was allowed to return to work.

*Comment.*—This patient was admitted with infiltration in his left lung. We were dealing with an acute, rapidly debilitating lesion. Lack of a positive diagnosis required a thoracotomy with resection.

After a successful pneumonectomy which, however, did not eradicate the fungus, complement-fixation and skin tests were negative. The negative skin test indicated little resistance to the disease.[10] Four months after surgery he developed a dissemination of the organism. All recognizable manifestations of this tragic occurrence have disappeared under the influence of undecylenic acid. Eight months after surgery the skin test is positive and the complement fixation test is negative. The patient is now working with no complaints. We will continue the treatment.

## DISCUSSION

In the past, surgery has not been used as a planned form of therapy for systemic blastomycosis with a unilateral pulmonary lesion. Collected figures show that less than 10 per cent of the systemic cases will live over five years. The survivors are usually in the group with a positive skin test and negative complement fixation test.

Our first patient with the negative skin test falls into the type of case with a poor prognosis. His case is, as far as we can determine, the first reported as treated by a planned pneumonectomy. Unfortunately, he did not survive.

The second patient, likewise with a negative skin test, fell into the 90 per cent of systemic cases not expected to survive. His diagnosis was made after excision of the primary lesion. Surgery did not prevent dissemination of disease, but it probably allowed development of some immunity, since his general condition improved in spite of postoperative jaundice.

Potassium iodide has been the only chemical available in the past. It has not prevented death in 90 per cent of the systemic cases. It has been of doubtful help in patients with a negative skin test. Schoenbach[11] recently reported one patient with systemic blastomycosis treated with intravenous stilbamidine. The patient is still under observation but the author thinks he is cured. He suggests that the diamidines be tried in severe yeastlike infection.

Undecylenic acid has been shown to inhibit growth of *Blastomyces dermatitidis* in vitro at concentrations of 10 mg. per cent. Two months' treatment with undecylenic acid eliminated the cutaneous form of blastomycosis in our second patient. The skin test is now positive and his general condition is good eight months after surgery. We can demonstrate no evidence of systemic disease. The drug in combination with surgery seems to have prevented certain death.

We will use undecylenic acid in the treatment of new systemic blastomycosis patients. Careful clinical studies will be made. We plan to do in vitro sensitivity studies using several different fatty acids and their salts against the most common pathogenic fungi.

If a pulmonary lesion is present, which would be suitable for resection, and the diagnosis is made without exploratory thoracotomy, prolonged medication will be given. If surgery does eventually become necessary, we hope to have protection against dissemination. We believe the drug will prove of great value in patients such as the one we have treated when the diagnosis is made from the surgical specimen. We plan to use up to 20 Gm. of undecylenic acid daily over long periods. The patient herein reported has been on 10.5 Gm. of undecylenic acid daily for four months without symptoms due to the drug.

## SUMMARY

1. Case histories of two patients with systemic blastomycosis have been presented. In each case the disease appeared localized to one lung. Each case was treated by pneumonectomy. We are not aware of similar case reports.

2. The first patient treated by planned pneumonectomy died six months after surgery. Iodide therapy seemed to have little effect on the spread in his left lung.

3. The diagnosis in the second patient was made after the lesion had been removed by pneumonectomy. A late spread occurred in spite of iodide therapy.

4. Disease in the second patient has either been controlled or cured by oral undecylenic acid.

5. We find no evidence that any of the fatty acids or their salts have been used to treat the deep mycotic infections.

We are indebted to Mr. Carl J. Hogue of the hospital laboratory section for his work with the fungus studies.

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# THE OPEN CLOSURE OF THE BRONCHUS AND THE RESECTION OF THE CARINA AND OF THE TRACHEAL WALL

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THE double lumen catheter constructed by Carlens for bronchospirometry and for one lung anesthesia has facilitated certain technical steps in lung resections to such a degree that it is now in daily use in our clinic.

The catheter* is made of rubber in two sizes. The lumen of each side corresponds to a circular area with a diameter of 7 mm. in the catheter for men and 6 mm. in the one for women. The total outer diameters are 13 and 11 mm., respectively.

The tube is introduced under topical anesthesia without the aid of fluoroscopy or bronchoscopy. Under mirror guidance it is introduced over a curved metal stylet. The rubber hook is tied down to the catheter with a moistened silk thread until the tube with a slight rotating movement has been passed through the larynx, when the slipknot is untied. The catheter is gently pushed downwards and turns automatically to the left when the curved stylet is withdrawn. During the introduction it exerts a slight pressure on the left tracheal wall and therefore enters the left main bronchus. It is then easy to feel when the hook is engaged by the carina. A slight pressure downwards is then exerted on the catheter when the cuffs are inflated. Then anesthesia and suction can be applied to each lung separately. The hook and the two rubber cuffs will maintain the catheter in correct position. It is not necessary nor advisable to have a higher pressure in the cuffs than enough to get an air-tight closure of the bronchus and the trachea. This is easily controlled when the artificial respiration is started.

It is also possible, either in general or in local anesthesia, to introduce the catheter over a straight metal stylet through a laryngoscope and remove the stylet and the silk thread as soon as the hook has passed the larynx. Many anesthetists prefer that technique, but in patients with a short neck and big teeth it may be of value to be familiar with the former procedure as well.

The experience from more than 300 resections has shown the main advantages of the flexible double lumen catheter for one lung anesthesia to be:

1. The open closure of the bronchus facilitating the resection of the carina and of the tracheal wall.

2. Prevention of transbronchial spread in wet cases and in tuberculosis making the "face-down" position unnecessary. Continuous suction can be applied to the bronchus draining the diseased area.

3. The lung can be deflated to provide more operating space when it is separated from the chest wall (see Fig. 2) and it can be inflated when lobar and intersegmental planes are developed (see Fig. 3).

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*The catheter described in this paper is obtainable from A. B. Stille-Werner, Stockholm 4, Sweden.

While points 2 and 3 have been described in an earlier paper, the advantage of the open closure will be discussed in this communication.

One lung anesthesia makes it possible to cut through the bronchus whenever this is suitable during the resection and to leave it open until it is convenient to close it. During the time the bronchus is left open the anesthetist leaves a small plastic catheter for continuous suction close to the cut edges of the bronchus to collect blood that may enter it.

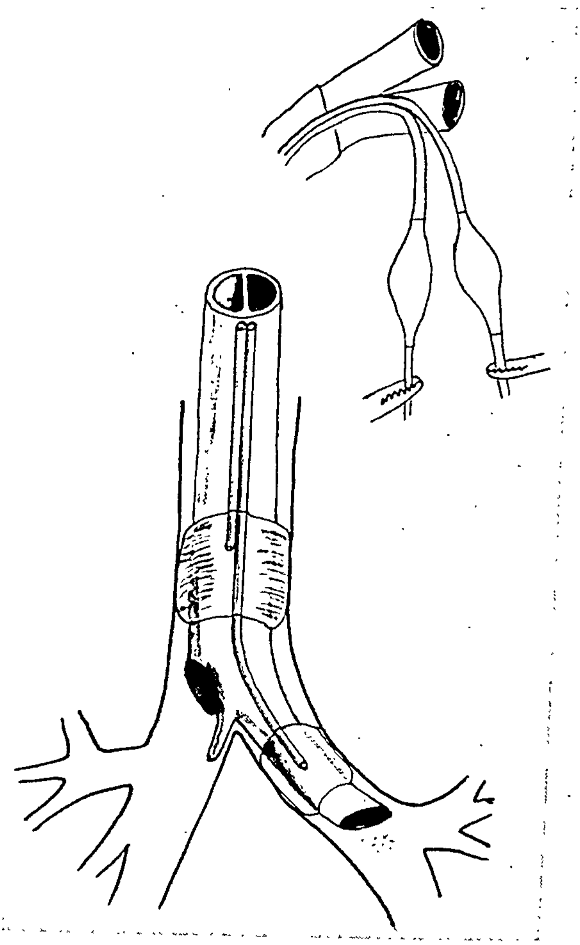

Fig. 1.—Diagram of the double lumen catheter in position. Notice the small rubber hook that is automatically engaged by the carina.

By this method it is not necessary to apply a clamp on the proximal part of the bronchus. If such a clamp is applied too firmly, it may injure the bronchial wall causing an impaired healing.

It may be possible to avoid the central bronchial clamp by other techniques, but the great advantage with one lung anesthesia is that there is no hurry in closing the bronchus. The bronchial tree can be inspected, bronchial anomalies may be detected and the correct interpretation of the bronchial anatomy may be confirmed in segmental resections. Bronchotomies and local excisions of tumors from the bronchial or tracheal wall have been much facilitated, see Case 3, Fig. 9. Through the open bronchus aspiration with a small rubber catheter may easily be carried out.

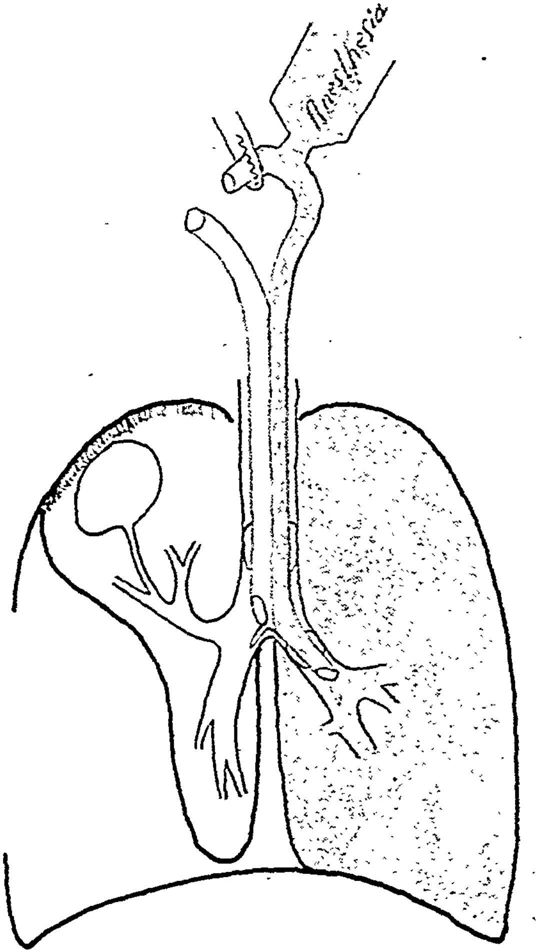

Fig. 2.—The lung can be deflated to provide more operating space when it is separated from the chest wall. Continuous suction is applied to the diseased lung and anesthesia to the healthy one.

*I. Bronchial Closure by Invagination.*—In pneumonectomies for tuberculosis without bronchial stenosis, the invagination technique according to Crafoord is preferred. The only danger of this technique is an obstruction of the proximal bronchus by the invaginated part. This may be avoided by a more exact estimation of the distance to the carina through the open bronchus. The invagination technique may also be performed in bilobectomies, but not in lobectomies or segmental resections when the bronchial stump is too short and a simple closure is preferred. Furthermore, there is no need for the use of this technique in cases with partial resections, where lung tissue is left behind covering the bronchial suture.

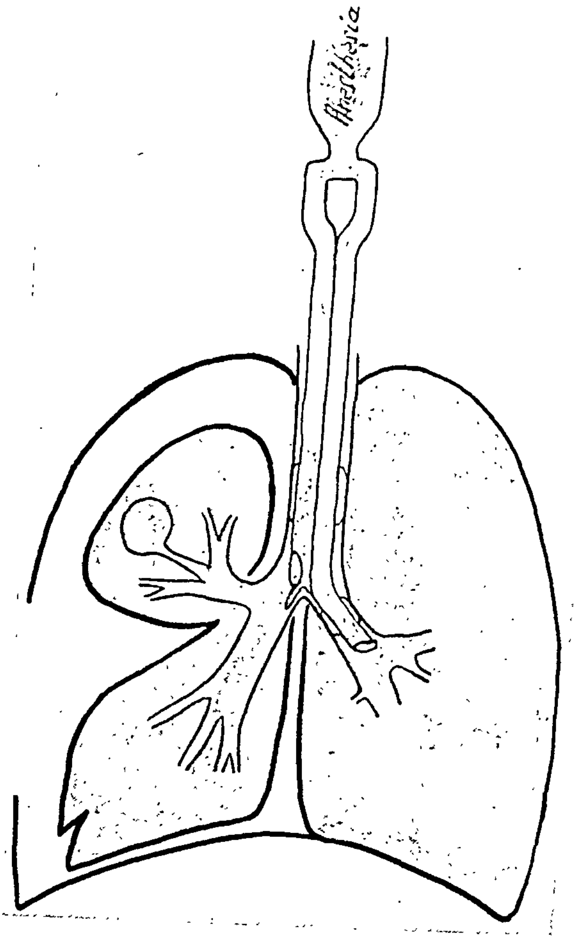

Fig. 3.—The lung can be inflated when lobar and intersegmental planes are developed.

The different steps of the invagination technique are: (1) The peripheral cartilaginous ring is exposed with a small knife and removed with a small elevator without injuring the adjacent external tissues or the thin submucosal layer (see Fig. 4*A*). This will facilitate the invagination considerably. (2) Four or five terminal interrupted sutures of No. 0000 Deknatel silk are placed. (3) A continuous number 0 catgut suture will render the bronchus airtight. (4) A number 2 silk purse-string suture is then placed around each corner (the corner constituting the border between the membranous and cartilaginous portion of the bronchial wall) (see Fig. 4*B*). (5) Each corner is then invaginated and two or three further interrupted silk sutures will complete the

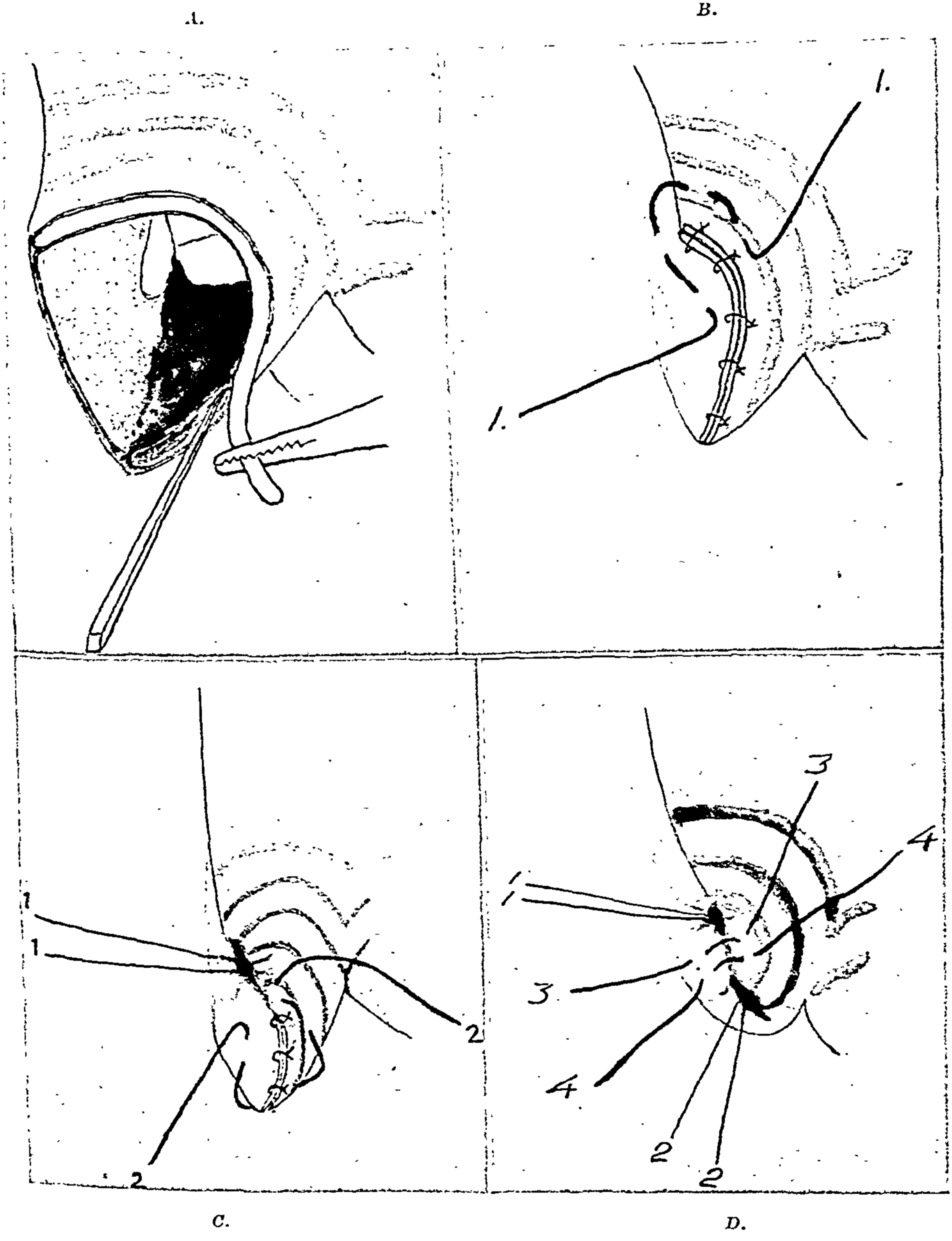

Fig. 4A.—The peripheral cartilaginous ring is removed by a small elevator. The rubber hook of the tube is riding over the carina.

Fig. 4B.—The first purse-string suture is placed (1-1).

Fig. 4C.—One corner has been invaginated and the second purse-string suture is placed (2-2).

Fig. 4D.—The invagination is completed by further interrupted silk sutures (3-3 and 4-4).

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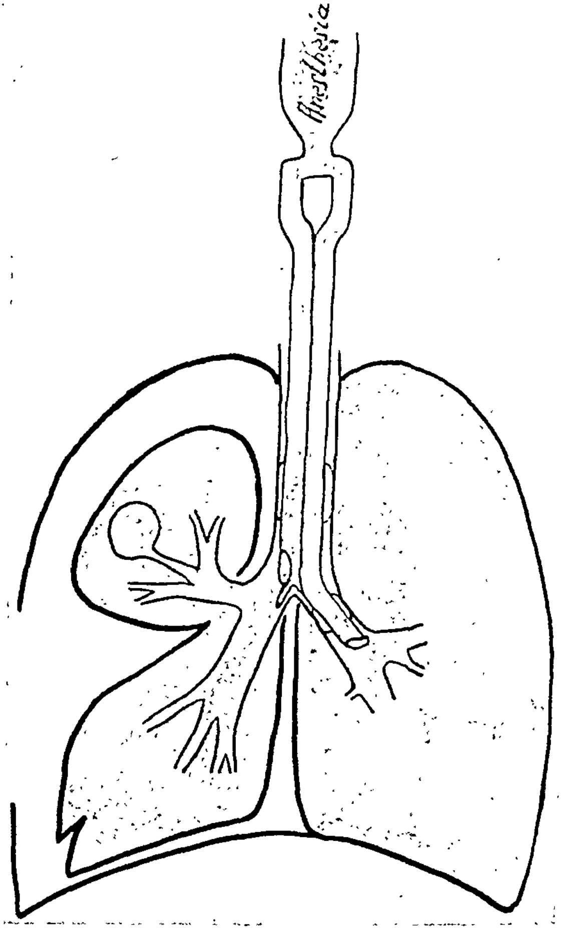

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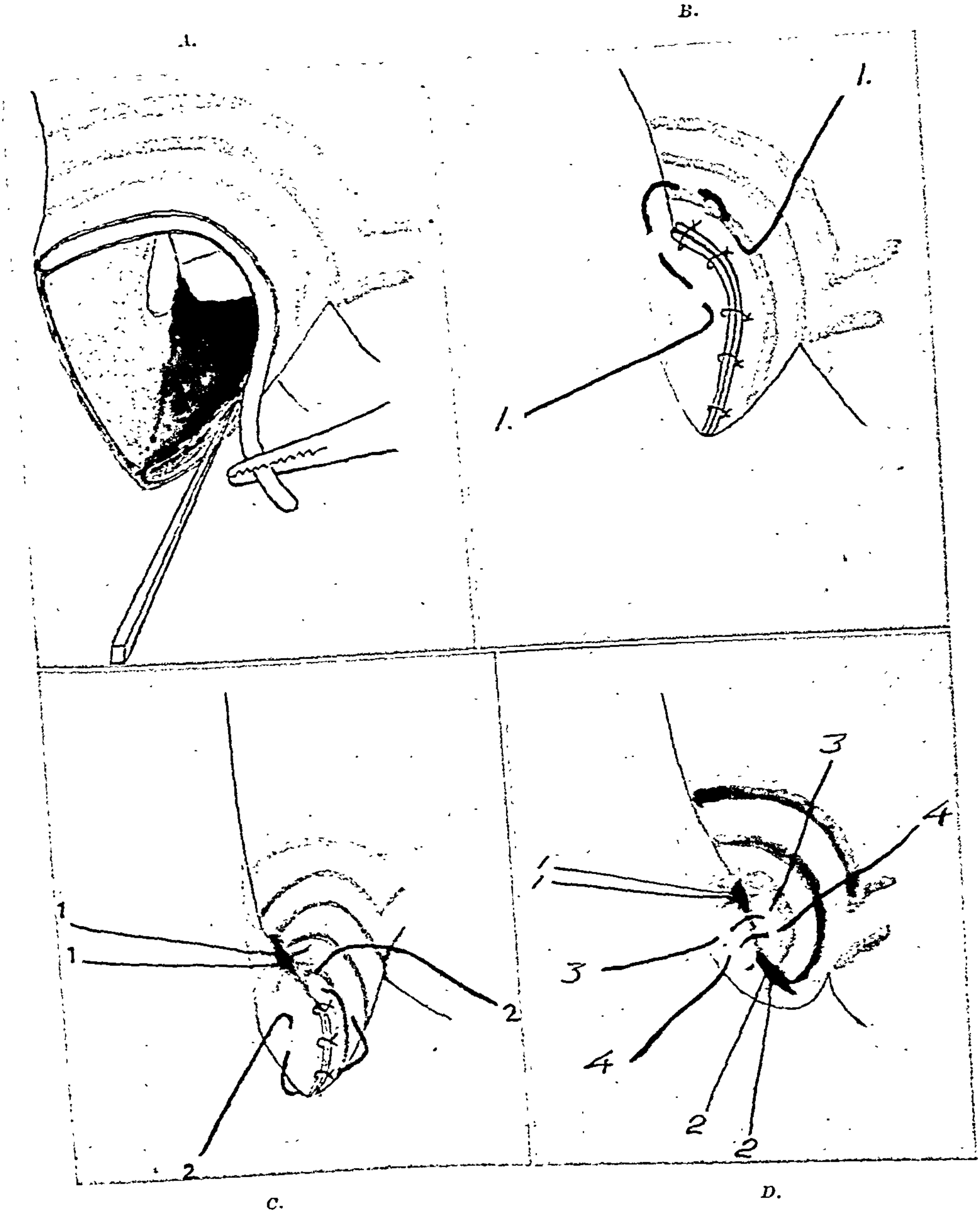

Fig. 4A.—The peripheral cartilaginous ring is removed by a small elevator. The rubber hook of the tube is riding over the carina.

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Fig. 4D.—The invagination is completed by further interrupted silk sutures (3-3 and 4-4).

invagination. Thus several millimeters of the external bronchial wall lie pressed together facilitating healing (see Fig. 4*E*). (6) Then a pedicled pleural flap is sewn over the bronchus.

*II. Simple Bronchial Closure.*—In neoplastic disease the most radical resection must be carried out. Thus the bronchus is often too short for invagination. With the aid of the double-lumen catheter the resection of the right lung, cutting out the bronchus from the trachea including carina and a portion of the left main bronchus can be performed at ease. This is illustrated in Case 1, Fig. 5.

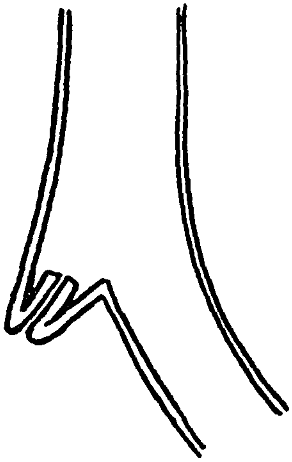

Fig. 4*E*.—Diagram of the invagination showing several millimeters of the external bronchial wall lie pressed together.

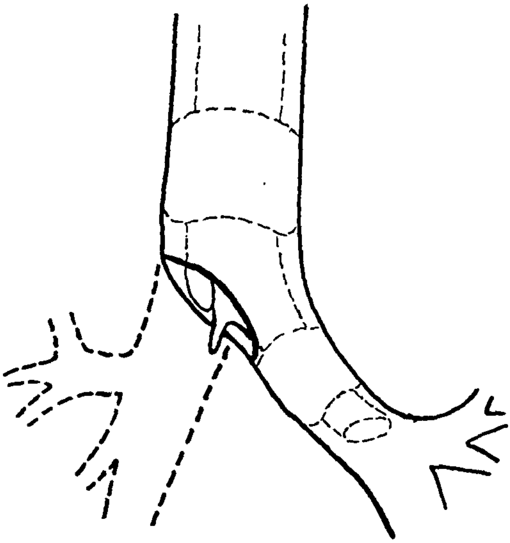

Fig. 5.—(Case 1.) High resection of right sided carcinoma including carina, part of trachea and the left main bronchus.

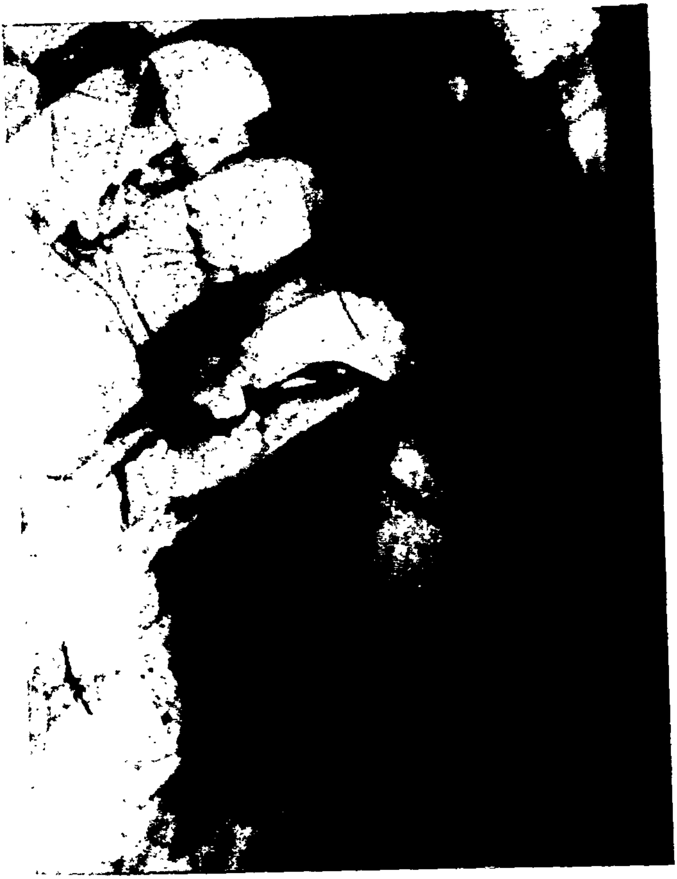

Fig. 6*A*.—(Case 2.) Bronchogram of the adenoma projecting into the trachea, obstructing the right and most of the left main bronchus. The abnormal right upper-lobe bronchus is seen originating from the trachea.

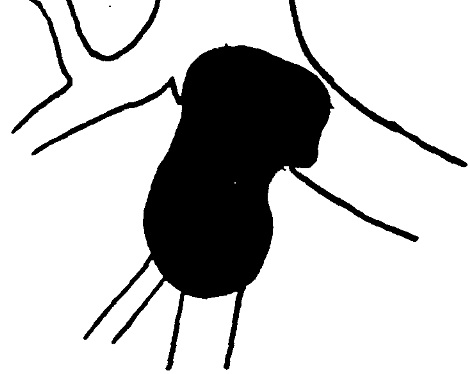

Fig. 6*B*.—Diagram of the adenoma in Case 2.

CASE 1.—A 50-year-old man was found to have a squamous-cell carcinoma of the right upper lobe, diagnosed at bronchoscopy. At operation a frozen section of the main bronchus close to the carina showed invasion of cancer. Then a portion of trachea and the carina with adjacent part of the left main bronchus was resected. Closure was then made with interrupted and a continuous terminal suture of fine braided stainless steel. A pleural flap was sewn over the suture. The postoperative course was uneventful.

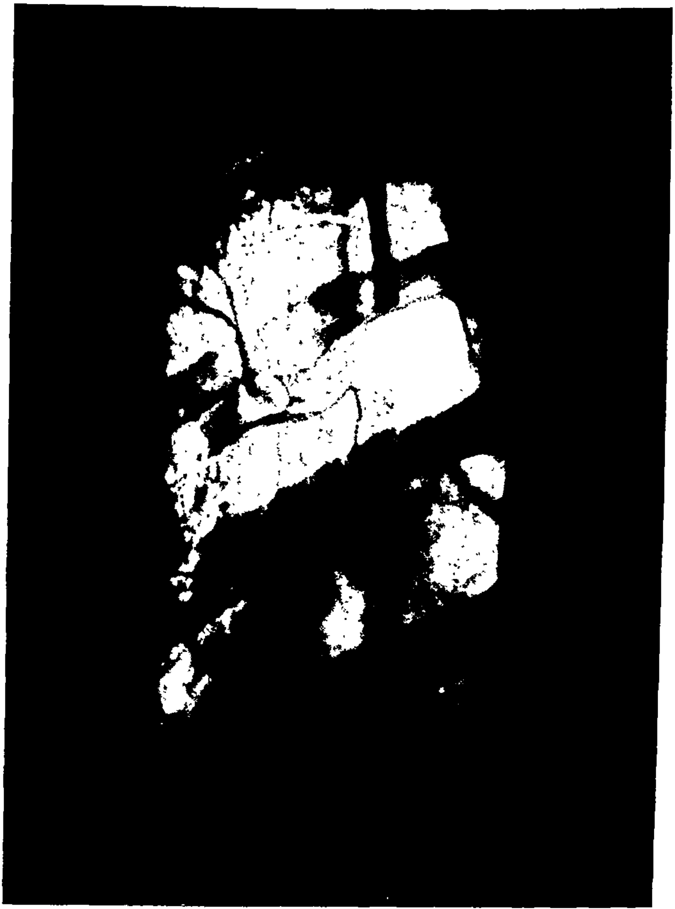

Fig. 7.—Bronchography after the preoperative endoscopic coagulation of the adenoma in Case 2.

In cases of bronchial adenoma the tumor with its extra- and intrabronchial portion can be cut out from the wall of the trachea or the bronchus and a plastic repair of the defect can be performed, see Cases 2 and 3.

CASE 2.—A 39-year-old woman was found to have a bronchial adenoma obstructing the right main bronchus and most of the left main bronchus. The patient was then breathing only with the right upper lobe through an abnormal bronchus originating in the trachea 2 cm. above the carina (see Fig. 6).

After thirteen endoscopic electrocoagulations both lungs were areated. Bronchography showed the origin of the tumor in the right main bronchus and bronchiectasis in the right middle and lower lobe (see Fig. 7).

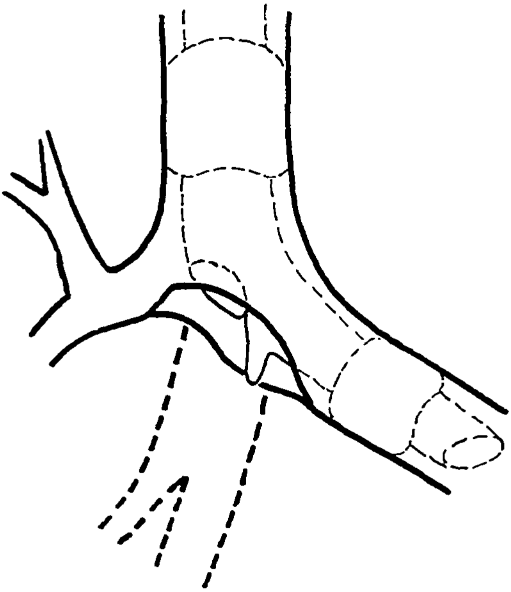

Fig. 8.—Diagram of the resection of the right lower and middle lobes including the carina and a small area of the left main bronchus in Case 2.

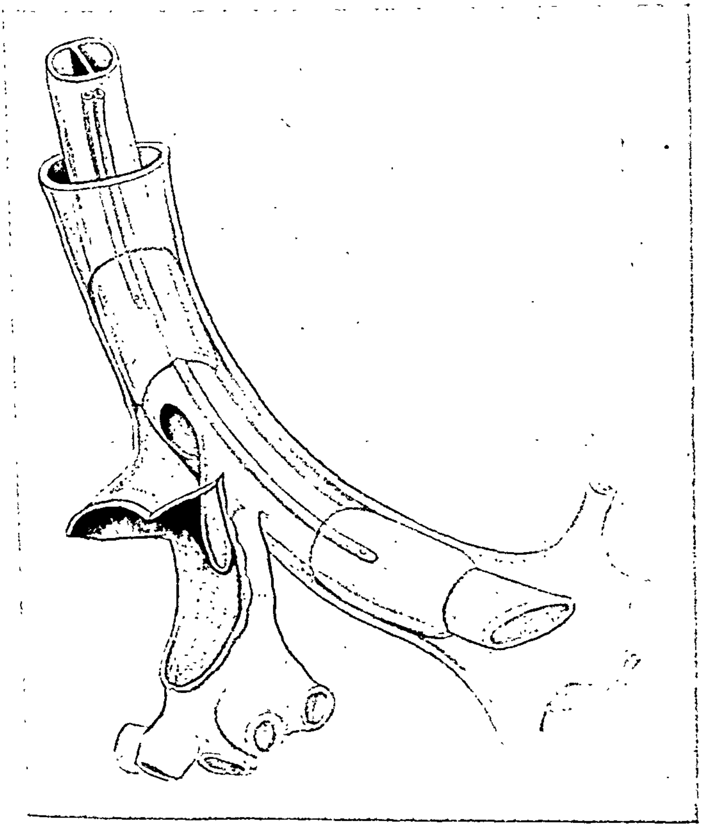

Fig. 9.—The right upper-lobe bronchus has been excised from the trachea.

At the operation the right lower and middle lobes were removed together with the carina and a small area of the left main bronchus (see Fig. 8). Closure was made by five interrupted and a continuous suture of fine braided stainless steel. The postoperative course was uneventful.

CASE 3.—A 44-year-old woman had a bronchial adenoma causing atelectasis of the right upper lobe. The right upper-lobe bronchus took its origin from the trachea at the level of the carina. At operation, the right upper lobe was removed, its bronchus being excised from the tracheal wall (see Fig. 9).

The technique of the simple bronchial closure is: (1) Dissection and removal of the peripheral bronchial cartilaginous ring. Cartilage has a poor blood supply and does not take part in healing. Sutures of braided stainless sutures of 0000 Deknatel silk on an atraumatic needle are placed. (3) Then a continuous suture of terminal 0000 Deknatel silk on a curved atraumatic needle is made. (4) A pedicled pleural flap is sewn over the bronchus.

Lobectomies and segmental resections may be performed in the same manner on the right as on the left side, but in cases of left pneumonectomies the described open closure of the bronchus cannot be carried out. When the lung is liberated and the hilar structures dissected, the cuffs are deflated and the catheter is withdrawn about 5 centimeters. Immediately beforehand the bronchial secretion on the left side is aspirated to prevent spread. Afterwards the proximal cuff only is reinflated, both lumina connected with the anesthesia machine and the catheter is used as a single lumen intratracheal tube. In the same moment as the catheter is withdrawn, a soft aortic clamp is placed at the origin of the bronchus.

## SUMMARY

One lung anesthesia makes it possible to cut through the bronchus whenever this is suitable during the resection and to leave it open until it is convenient to close it. It is not necessary to apply a clamp on the proximal part of the bronchus. Resection of the carina with adjacent part of trachea and left main bronchus is greatly facilitated.

## REFERENCES

Björk, V. O., and Carlens, E.: The Prevention of Spread During Pulmonary Resection by the Use of a Double Lumen Catheter, J. THORACIC SURG. 20: 151, 1950.

Crafoord, C.: On the Technic of Pneumonectomy in Man, Acta Chir. Scandinav. Supp. 54: 81: 1, 1938.

Jones, J. C.: Closure of the Bronchus in Pulmonary Resection, Am. Rev. Tuberc. 51: 55, 1945.

Rienhoff, W. F., Gannon, J., and Sherman, I.: Closure of the Bronchus Following Total Pneumonectomy: Experimental and Clinical Observations, Ann. Surg. 116: 481, 1942.

Sweet, R. H.: Closure of the Bronchial Stump Following Lobectomy or Pneumonectomy, Surgery 18: 82, 1945.

# THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

## THIRTY-SECOND ANNUAL MEETING

MAY 8, 9, AND 10, 1952, BAKER HOTEL
DALLAS, TEXAS

## PROGRAM

*Thursday Morning, May 8, 1952*

9:00 A.M. BUSINESS MEETING

9:30 A.M. SCIENTIFIC SESSION

1. **Experimental Methods for Detection of Changes of Blood Supply to the Heart.**
   Alfred Goldman, Clinton Shaw (by invitation), Eliot Corday (by invitation), Joshua Fields (by invitation), S. Rexford Kennamer (by invitation), Allen Smith (by invitation), Inga Lindgren (by invitation), and Myron Prinzmetal (by invitation), Los Angeles, Calif.
2. **Artificial Oxygenation and Circulation During Complete Bypass of the Heart.**
   James A. Helmsworth. Roger T. Sherman (by invitation), Samuel Kaplan (by invitation) and Leland C. Clark (by invitation), Cincinnati, Ohio
3. **Some Physiological Aspects of the Artificial Heart Problem.**
   F. D. Dodrill, Edward Hill (by invitation) and Robert Gerisch (by invitation), Detroit, Mich.
4. **Technique of Producing Mitral Stenosis of Controlled Degree.**
   Robert G. Ellison, Robert C. Major, Raymond W. Pickering (by invitation) and Wm. F. Hamilton (by invitation), Augusta, Ga.
5. **Surgical Approach for Stenotic Lesions of the Semilunar Valves by Excision and Cusp Replacement under Direct Vision.**
   Robert S. Litwak (by invitation), and Howard L. Gadboys (by invitation), Boston, Mass.
6. **Experimental Pulmonary Venous Occlusion.**
   C. Rollins Hanlon, David Sabiston (by invitation), and Donald R. Burke (by invitation), St. Louis, Mo.

*Thursday Afternoon, May 8, 1952*

2:00 P.M. SCIENTIFIC SESSION

7. **Localized Fibrous Mesothelioma of the Pleura.**
   O. Theron Clagett, John R. McDonald (by invitation), and Herbert W. Schmidt (by invitation), Rochester, Minn.
8. **Resection of the Trachea for Carcinoma—Report of Two Cases.**
   Bert H. Cotton and Joao R. F. Penido (by invitation), Beverly Hills, Calif.
9. **The Surgical Treatment of Carcinoma Involving the Hypopharynx, Larynx, and Upper Esophagus.**
   Orville F. Grimes (by invitation) and H. Brodie Stephens, San Francisco, Calif.
10. **A Consideration of Palliative Treatment with Direct Attacks upon Carcinoma of the Esophagus.**
    Mark M. Ravitch, Henry T. Bahnson (by invitation) and Thomas N. P. Johns (by invitation), Baltimore, Md.

11. **A Pathologic Study of Large Tissue Sections of Bronchogenic Carcinoma.**
Edward J. McGrath and Edward A. Gall (by invitation), Cincinnati, Ohio
12. **The Use of Streptokinase-Streptodornase (Varidase) in the Management of Early Postoperative Partial Pulmonary Resections.**
C. Thomas Read (by invitation), Phoenix, Ariz.

*Friday Morning, May 9, 1952*

9:00 A.M. SCIENTIFIC SESSION

13. **Traumatic Aneurysm of the Thoracic Aorta.**
R. K. Hollingsworth and W. Johnston (by invitation), Martinsburg, West Virginia
14. **Healing of Extensive Cardiac Wounds.**
C. G. Thomas, Jr. (by invitation), Chapel Hill, N. C. and S. E. Ziffren (by invitation), Iowa City, Iowa
15. **Traumatic Hemopericardium and Constrictive Pericarditis.**
Johann L. Ehrenhaft and Rodman E. Taber, Iowa City, Iowa.
16. **Rupture of the Esophagus.**
Robert A. Anderson (by invitation), Tulsa, Oklahoma and George M. Curtis, Columbus, Ohio
17. **The Effects of Atelectasis on Pulmonary Arterial Blood Flow in Dogs.**
Richard M. Peters (by invitation) and Albert Roos (by invitation), St. Louis, Mo.
18. **Surgical Management of Pulmonary Emphysema.**
Gerald L. Crenshaw (by invitation) and Donald F. Rowles (by invitation), Oakland, Calif.

*Friday Afternoon, May 9, 1952*

2:00 P.M. EXECUTIVE SESSION

3:00 P.M. SCIENTIFIC SESSION

**Address of the President—**Frank B. Berry, New York, N. Y.

19. **The Care of Thoracic and Thoracoabdominal Wounds in the Combat Zone in Korea.**
Major Edward Ernest Rocky, M.C. (by invitation), A.P.O. San Francisco, Calif.
20. **War Wounds of the Chest: Experiences with 1,535 Korean Casualities in an Army Hospital Overseas.**
Major A. R. Valle, M.C. (by invitation), Tokyo

6:30 P.M. COCKTAIL PARTY—Baker Hotel.

*Saturday Morning, May 10, 1952*

9:00 A.M. SCIENTIFIC SESSION

21. **Silicotic and Tuberculosilicotic Lesions Simulating Bronchogenic Carcinoma.**
Frederick G. Kergin, Toronto, Ont.
22. **The Elective Resection of Nodular Tuberculosis.**
D. O. Shields (by invitation) and John S. Chapman (by invitation), Dallas, Texas
23. **The Complications and the Results of Treatment of Bronchopleural Fistula Following Resection for Tuberculosis.**
James D. Murphy, Barney B. Becker (by invitation) and H. V. Swindell (by invitation), Oteen, N. C.
24. **Plastic Sponge Prosthesis Following Resection in Pulmonary Tuberculosis.**
Joseph W. Gale and Anthony R. Curreri, Madison, Wis.

25. The Role of the Inferior Esophageal Constrictor in the Production of Lower Esophageal Disease.
    Earle B. Kay, Cleveland, Ohio
26. The Delayed Restoration of Oral Alimentation in Children with an Esophageal Defect Remaining after Closure of a Tracheo-Esophageal Fistula.
    N. Logan Leven and Richard L. Varco, St. Paul, Minn.

*Saturday Afternoon, May 10, 1952*

2:00 P.M. SCIENTIFIC SESSION

27. Surgical Treatment for Congenital Aortico-Pulmonary Fistula: Experimental and Clinical Aspects.
    H. William Scott, Jr., and David C. Sabiston (by invitation), Baltimore, Md.
28. Pulmonary Function after Bilateral Multiple Segmental Resection for Bronchiectasis.
    Richard H. Overholt, Benjamin Etsten (by invitation) and James H. Walker (by invitation), Boston, Mass.
29. The Indications For and the Results of Commissurotomy for Mitral Stenosis.
    Robert P. Glover, Thomas J. E. O'Neill, James M. Harris (by invitation) and O. Henry Janton (by invitation), Philadelphia, Pa.
30. The Cure of Two Aneurysms Associated with Patent Ductus with a Résumé of Seventy Cases of Patent Ductus Surgically Treated.
    Emile Holman and Frank Gerbode, San Francisco, Calif.
31. Anatomical (Histological) Basis and Efficient Clinical Surgical Techniques for the Restoration of the Coronary Circulation.
    Charles P. Bailey, R. C. Truex (by invitation), George Geckeler (by invitation) and Nicholas Antonius (by invitation), Philadelphia, Pa.
32. Pulmonary Valvulotomy: Description of a New Operative Approach with Comments about Diagnostic Characteristics.
    Harris B. Shumacker, Jr., Indianapolis, Ind.

## PAPERS TO BE READ BY TITLE

1. The Use of Viomycin with Excisional Surgery for Pulmonary Tuberculosis.
   H. E. Walkup (by invitation), James D. Murphy (by invitation) and Oscar Kanner (by invitation), Oteen, N. C.
2. Proposed Clinical Staging of Bronchogenic Carcinoma.
   F. X. Byron, Los Angeles, Calif.
3. Trans-Cervical Superior Mediastinal Biopsy.
   Ralph Friedlander and Benjamin Burbank, Brooklyn, N. Y.
4. Thoracoplasty and Resection for Pulmonary Tuberculosis: Follow-up of 229 Patients Treated by Posterior Thoracoplasty, by Resection or by Thoracoplasty and Resection.
   F. A. Hughes, Jr., C. C. Lowry (by invitation) and J. W. Polk (by invitation), Memphis, Tenn.
5. The Results of Thoracoplasty for Pulmonary Tuberculosis Eleven to Sixteen Years after Operation.
   Arthur H. Aufses and Maurice S. Harte (by invitation), New York, N. Y.
6. Personal Experiences with 10 Consecutive Heart Wounds Treated Successfully by Pericardiotomy and Cardiorrhaphy.
   Emil A. Naclerio (by invitation), Aubre De L. Maynard (by invitation) and John W. V. Cordice (by invitation), New York, N. Y.
7. Simultaneous Resection and Thoracoplasty for Tuberculosis.
   James E. Dailey, Houston, Texas

8. **Pulmonary Tuberculosis—A Comprehensive Analysis of 470 Cases of Excisional Surgery Operated upon at Sea View Hospital Between 1944 and 1951.**
   Louis R. Davidson, Hyman Alexander (by invitation), Bernard J. Kesner (by invitation) and Gerald J. Lustig (by invitation), New York, N. Y.
9. **Pulmonary Diestention as Seen in the Lordotic Roentgenogram.**
   Joseph Gordon and W. Berkeley Zinn (by invitation), Ray Brook, N. Y.
10. **Intrapulmonary Hematoma.**
   Colonel John M. Salyer (by invitation), Lt. Col. Robert C. Major, Major Hu A. Blake (by invitation) and Col. James H. Forsee, Denver, Colo.
11. **Chest Wall Pain.**
   E. K. Johnson and J. L. Mangiardi (by invitation), Brooklyn, N. Y.
12. **The Use of Suture Constriction of the Upper Lobe in the Treatment of Pulmonary Tuberculosis (Paulino Procedure).**
   Paul T. DeCamp, John W. Overstreet (by invitation), Thomas G. Baffes (by invitation) and Alton Ochsner, New Orleans, La.
13. **The Diagnosis of Functionally Severe Patent Ductus Arteriosus in Infancy and Early Childhood.**
   F. Mason Sones, Jr. (by invitation) and Donald B. Effler, Cleveland, Ohio.
14. **Major Thoracic Surgery in Patients over Seventy.**
   William L. Watson, William G. Cahan (by invitation) and Dorothy P. Reaves (by invitation), New York, N. Y.
15. **Value of Scalene Node Biopsy.**
   Lawrence M. Shefts, San Antonio, Texas
16. **The Present Status of Bronchography with Absorbable Water-Soluble Media.**
   Mordant E. Pack (by invitation), A. J. Neerken (by invitation) and Emanuel Salzman (by invitation), Denver, Colo.

The Journal of
Thoracic Surgery

VOL. 23 MAY, 1952 NO. 5

# Original Communications

## AN ANATOMIC SURVEY OF ANOMALOUS PULMONARY VEINS: THEIR CLINICAL SIGNIFICANCE

JOHN E. HEALEY, JR., M.D.
PHILADELPHIA, PA.

IT IS only in recent years that attention has been focused on the anatomic variations and anomalies of certain thoracic structures. This increased interest in the variational anatomy of the thorax is but a sequel to the great advances that have been made in the field of thoracic surgery. Thus, of the total number of anomalous pulmonary veins found recorded in the literature, fully 43 per cent have been recorded within the last twenty years. In 1942, Brody[5] presented an excellent review of the literature on the abnormal connections between the pulmonic and systemic venous systems. Beginning his review with the first case reported by Winslow[22] in 1739, he collected from the literature a total of 106 cases. In 1944, Hughes and Rumore[14] cited seven additional cases which were not listed by Brody in his survey. In our own survey, we found nine other cases reported in the literature prior to 1942 and twenty-two cases which have been reported since then. Adding all cases reported in the literature to the three cases presented in this paper brings the total number of anomalous pulmonary-systemic connections thus far recorded to 147 cases.

In view of the existence of other anatomic variations, it is quite obvious that the incidence of anomalous pulmonary veins is actually higher than that which is suggested by the relatively few cases (147) reported over a period of two hundred years. Undoubtedly many additional cases have been observed during the course of two centuries, but because of lack of interest were not reported. The significance of aberrant pulmonary-systemic connections has rarely been emphasized in the past. Variations in the number of pulmonary veins occurred with such frequency that they did not merit mention in the literature, except when they were accompanied by an anomalous drainage of one or more of the veins. As far as we could ascertain, a statistical analysis of the variations of the pulmonary veins in a large series of cases has thus far not

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From the Daniel Baugh Institute of Anatomy, Jefferson Medical College.
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been made. Therefore, one cannot come to a definite conclusion as to their incidence. The *purpose of this paper is to present our observations in the* dissection of 251 adult cadavers, to correlate these findings with pertinent data attainable from the literature, and briefly to review the clinical significance of anatomic variations of the pulmonary veins.

Poirier and Charpy[19] classified the variations of the pulmonary veins into two types: (1) abnormal number of pulmonary veins, and (2) abnormal drainage of the pulmonary veins, i.e., abnormal connections between the pulmonic and the systemic venous systems. The latter were subdivided into (a) complete drainage, and (b) incomplete drainage of the pulmonary circuit. In this paper Charpy's classification is retained but is modified as follows:

I. Abnormal number of pulmonary veins
  a. Decrease in number
    1. Normal drainage into left side of the heart
    2. Abnormal drainage into right side of the heart
  b. Increase in number
    1. Normal drainage into left side of the heart
    2. Abnormal drainage into the right side of the heart
    3. Mixed drainage
      a. Into left side of the heart
      b. Into right side of the heart
II. Abnormal drainage of the pulmonary veins
  a. Complete drainage of the pulmonary circuit into the right side of the heart
  b. Incomplete drainage of the pulmonary circuit into the right side of the heart

### ABNORMAL NUMBER OF PULMONARY VEINS

Only a few statistical reports on the variations of the number of pulmonary veins can be found in the literature. In textbooks of anatomy, all authors describe two left and two right pulmonary veins emptying into the left atrium. Many variations are possible, and undoubtedly occur more frequently than is generally known.

The most common variation observed is the presence of a common pulmonary vein on either the right or left side (Fig. 1). *Adachi,*[1] *in an examination of* ninety-one cadavers, encountered eleven cases (12.1 per cent) having a common pulmonary vein; whereas, Healey and Gibbon[13] found fifty-two cases in 185 adult cadavers. Since publication of the latter report we have found eight additional cases in sixty-six bodies. Adding the cited figure given by Healey and Gibbon gives a total of sixty cases in 251 specimens (23.9 per cent) in which a common pulmonary vein was present. The three cases with a common vessel as described by Brantigan[4] do not lend themselves to a statistical study, for they were observed in an unspecified number of operative procedures. In each of the above described specimens the common pulmonary vein drained into the left atrium. However, there have been thirty cases reported in which a common pulmonary vein emerging from either lung drained into the right side of the heart, either directly or indirectly.

A common pulmonary vein may then be present on either the right or left side, and may drain into either the right or left atrium. The earlier writers, such as Poirier and Charpy[19] state that such a common vein is not at all uncommon on the left side. Our dissections confirm this statement, for in 251 specimens we found fifty-three such vessels on the left side and only seven on the right side. The three cases mentioned by Brantigan occurred likewise on the left side. However, of the eleven common pulmonary veins described by Adachi, six were on the right side and five were on the left side. Of the thirty cases surveyed in the literature, each with an abnormal site of drainage, twenty were on the right side and ten on the left.

The next most common variation of the pulmonary veins is an increase in their number. There may be three veins from either lung entering the left atrium, but again few such cases have been reported. In our dissections this variation occurred more frequently on the right side, a separate vein being present for each lobe (Fig. 2). The additional vessel when present is known as the middle pulmonary vein. Adachi found three such vessels on the right side in 150 dissections (2.0 per cent). We have seen four right middle pulmonary veins in 251 bodies examined, an incidence of 1.6 per cent. Brantigan described two cases observed at operation in which three pulmonary veins were present on the right side and emptied into the left atrium. Four other cases have been reported in the literature in which three veins were present on the right side, but in each of these specimens at least one of the veins drained aberrantly. As regards the left side, Adachi encountered three separate pulmonary veins on two occasions while examining eighty-nine bodies (2.2 per cent). A specimen with three left pulmonary veins entering the left atrium was not observed in our dissections.

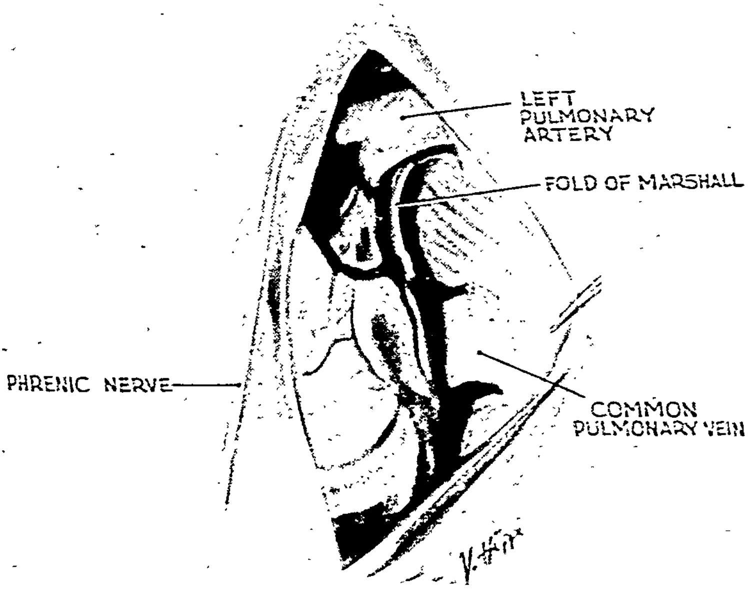

Fig. 1.—Left common pulmonary vein. (Reproduced from J. THORACIC SURG. 19: 564-574, 1950.)

When more than three veins are present on one side, one or more of them, in most instances, exhibit an anomalous drainage. Such cases are relatively rare. Atkinson and associates[3] described a case in which four right pulmonary veins were present, two of which drained into the superior vena cava. Didion[10] encountered a case in which four veins were present on the right side, but all of these entered the right atrium. Two cases have been reported in the literature in which five veins drained the right lung, and in both of these cases four of the veins drained into an abnormal site. Hyrtl[16] described a case in which the left upper lobe was drained by six veins, each vein emptying into a persistent left superior vena cava.

The variations in the number of pulmonary veins are easily explained on the basis of embryologic development. Primitively, a single pulmonary vein drains both lungs. With the growth of the left atrium it becomes absorbed into the latter; so that at first one, then two, and finally four pulmonary veins empty into the left side of the heart. Should less than the normal amount of absorption take place, then a common pulmonary vein will be formed on one side or on the other. If more than the usual amount of absorption takes place, there will be an increase in the number of pulmonary veins.

### ABNORMAL DRAINAGE OF THE PULMONARY VEINS

The entire pulmonary circuit may drain into the right side of the heart, either directly or indirectly. Sixty-one such cases have been reported in the literature. The most frequent sites of drainage of these anomalous vessels are shown in Table I. The average age of all the reported cases is 1.8 years; however, four of these survived beyond the age of 10 years, the oldest being 27 years. Excluding these four cases, the average age drops abruptly to 5.8 months. Survival in these cases is only possible if some form of communication between the right and left sides of the heart persists. Of the sixty-one cases, eleven lived beyond the age of 1 year. In the eleven cases, six had a patency of the foramen ovale, two had a patency of the ductus arteriosus, two had a single atrium, and one had an interatrial septal defect.

TABLE I. SITES OF DRAINAGE OF ANOMALOUS PULMONARY VEINS

| COMPLETE DRAINAGE (61 CASES) | NO. | PER CENT | INCOMPLETE DRAINAGE (86 CASES) | NO. | PER CENT |
|---|---|---|---|---|---|
| Superior vena cava | 10 | 15.9 | Superior vena cava | 33 | 35.8 |
| Coronary sinus | 10 | 15.9 | Left innominate vein | 19 | 20.6 |
| Right atrium | 10 | 15.9 | Right atrium | 18 | 19.6 |
| Portal vein | 9 | 14.3 | Left superior vena cava | 7 | 7.6 |
| Left innominate vein | 8 | 12.7 | Inferior vena cava | 4 | 4.3 |
| Left superior vena cava | 7 | 11.1 | Azygos vein | 3 | 3.3 |
| Ductus venosus | 3 | 4.7 | Coronary sinus | 2 | 2.2 |
| Right innominate vein | 2 | 3.1 | Single atrium | 2 | 2.2 |
| Single atrium | 1 | 1.6 | Left subclavian vein | 1 | 1.1 |
| Left subclavian vein | 1 | 1.6 | Portal vein | 1 | 1.1 |
| Azygos vein | 1 | 1.6 | Left ventricle | 1 | 1.1 |
| Sinus venosus | 1 | 1.6 | Thoracic duct | 1 | 1.1 |
| Total | 63* | 100.0 | Total | 92† | 100.0 |

*Two cases had two different sites of drainage.
†Six cases had two different sites of drainage.

There may be an incomplete drainage of the pulmonary circuit into the systemic venous system, and of this type eighty-six cases have been reported, including the three reported in this paper. These cases differed both in regard to the portion of the lung drained, and to the site of entrance of the anomalous vessel. In thirty-three of the eighty-six cases complete drainage of one lung was present (the right lung in twenty-two and the left lung in eleven instances). Anomalous drainage of a portion of one lung has been reported in regard to all lobes except the left lower lobe. The most frequent lobes involved were the right upper lobe (thiry-three cases) and the left upper lobe (twenty-two cases). The most frequent sites of drainage were into the superior vena cava (thirty-three cases), left innominate vein (nineteen cases), and the right atrium (eighteen cases) (Table I). Of the seventy cases in which the age was recorded, 77 per cent had reached maturity.

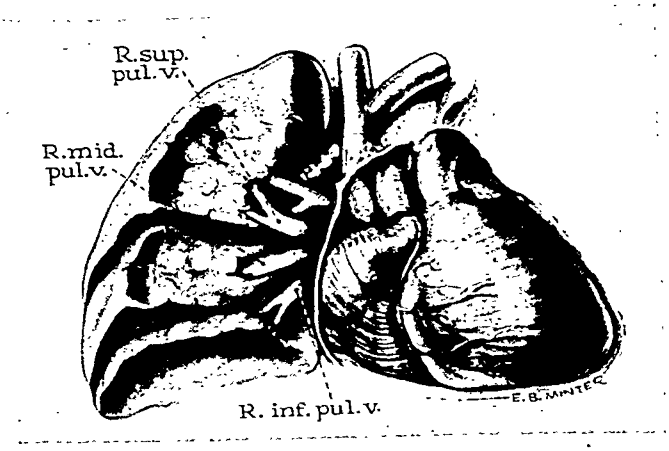

Fig. 2.—Three right pulmonary veins.

TABLE II. INCIDENCE OF ANOMALOUS PULMONARY-SYSTEMIC CONNECTIONS

| AUTHOR | TOTAL BODIES (NUMBER) | ANOMALOUS VESSELS (NUMBER) | PER CENT |
|---|---|---|---|
| Adachi | 270 | 2 | 0.7 |
| Hughes & Rumore | 280 | 2 | 0.7 |
| Healey & Gibbon | 251 | 1 | 0.4 |
| Total | 801 | 5 | 0.62 |

Adachi encountered two cases with a partial drainage of the pulmonary circuit into the systemic system in 270 cadavers. Hughes and Rumore described two cases in 280 dissections, an incidence of 0.7 per cent in both series (Table II). In our studies of 251 bodies we found one instance (0.4 per cent) of an anomalous drainage of the pulmonary veins. Since then we have observed two additional cases. A report on the three cases observed may be briefly outlined as follows:

CASE 1.—*The anomalous pulmonary drainage occurred in a white man 65 years of age whose clinical history was unobtainable and the cause of death unknown. Upon opening the thorax, a large vein was seen ascending in the superior mediastinum. This vein received tributaries from all segments of the left upper lobe including the lingular division. It ascended over the anterior aspect of the left pulmonary artery, lying parallel with the ductus arteriosus; thence it ran over the anterior surface of the aortic arch and terminated by emptying into the left innominate vein 3.5 cm. from its entrance into the superior vena cava. As a trunk, the anomalous vein measured 3.2 cm. in circumference (Fig. 3).

The left inferior pulmonary vein which drained the lower lobe emptied normally into the left atrium. Aside from the presence of a patent ductus arteriosus which measured 1.4 cm. in circumference, no other congenital abnormalities of the heart could be distinguished.

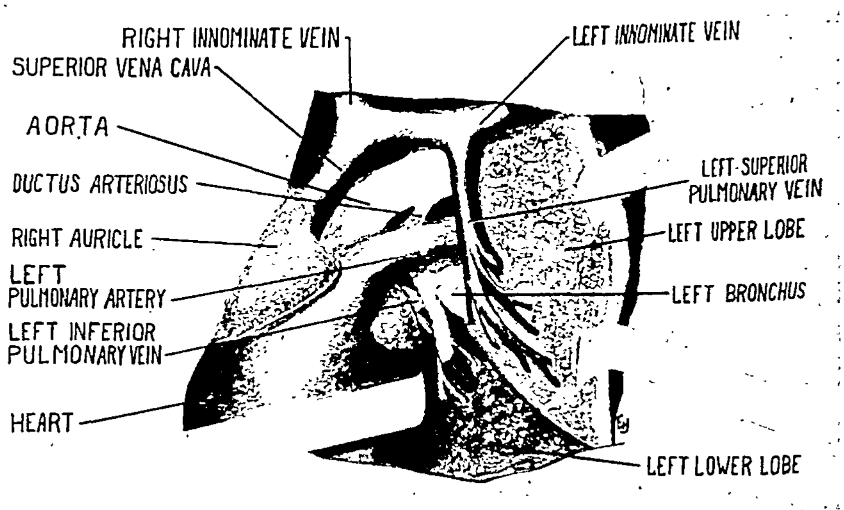

Fig. 3.—Anomalous vessel found in a dissected specimen. Left superior pulmonary vein drains into the left innominate vein. The left inferior pulmonary vein follows its normal path of drainage into the left atrium. (Reproduced from J. THORACIC SURG. 19: 864-874, 1950.)

CASE 2.—The anomalous vessel in this case was found in a 40-year-old white man. It drained the entire left upper lobe and coursed cephalad anterior to the bronchus, left pulmonary artery, and arch of the aorta. It terminated in the left innominate vein 5 cm. from the point where the latter joined the superior vena cava, and measured 4.8 cm. in circumference (Fig. 4).

The left inferior pulmonary vein was present and drained into the left atrium. The ductus arteriosus and foramen ovale were both completely sealed off.

CASE 3.—The following case of an anomalous drainage was seen in an adult male cadaver. The left upper lobe was drained by a left superior pulmonary vein, but instead of emptying into its normal site this vessel took an aberrant course, ascending into the superior mediastinum. In this part of its course it was joined by two vessels; (a) the superior intercostal vein of the left side, and (b) a vein which coursed anterior to the pulmonary artery and extended caudad into the vestigial fold of Marshall. This latter vein, however, was not continuous below with the oblique vein of the left atrium, and therefore, was not a complete persistence of the left superior vena cava. The three vessels observed, namely the left superior intercostal, the remnant of the left superior vena cava, and the left superior pulmonary vein joined to form a single trunk which entered the left innominate vein (Fig. 5).

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*This case was briefly mentioned by Healey and Gibbon in J. THORACIC SURG. 19: 864, 1950.

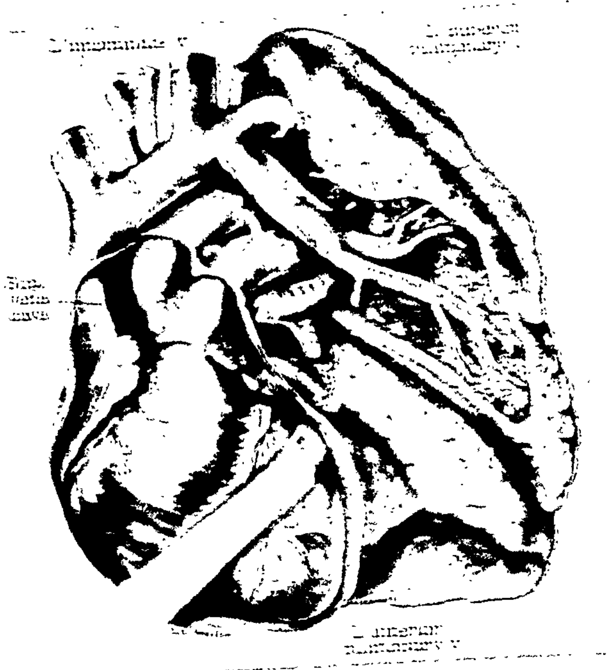

Fig. 4.—An anomalous pulmonary-systemic connection with the left superior pulmonary vein draining into the left innominate vein (white man, aged [illegible] years).

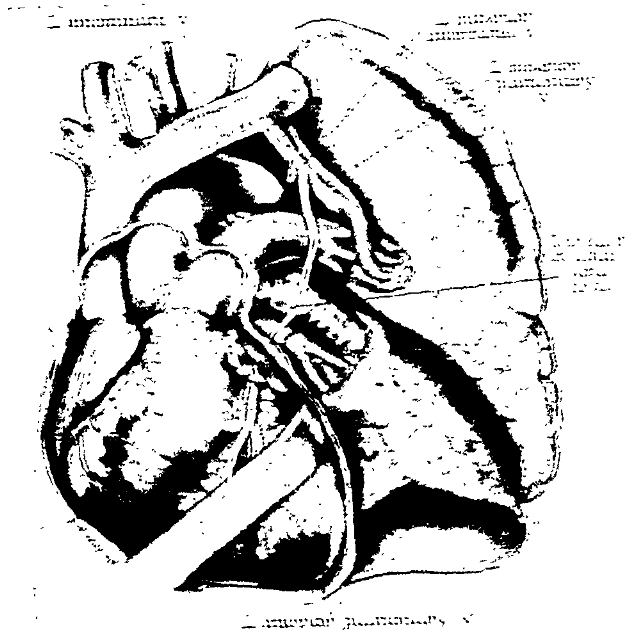

Fig. 5.—Drainage of the left upper lobe into the left innominate vein. Note the junction of the left superior intercostal vein and of the remnant of the left superior vena cava with the left superior pulmonary vein.

CASE 1.—*The anomalous pulmonary drainage occurred in a white man 65 years of age whose clinical history was unobtainable and the cause of death unknown. Upon opening the thorax, a large vein was seen ascending in the superior mediastinum. This vein received tributaries from all segments of the left upper lobe including the lingular division. It ascended over the anterior aspect of the left pulmonary artery, lying parallel with the ductus arteriosus; thence it ran over the anterior surface of the aortic arch and terminated by emptying into the left innominate vein 3.5 cm. from its entrance into the superior vena cava. As a trunk, the anomalous vein measured 3.2 cm. in circumference (Fig. 3).

The left inferior pulmonary vein which drained the lower lobe emptied normally into the left atrium. Aside from the presence of a patent ductus arteriosus which measured 1.4 cm. in circumference, no other congenital abnormalities of the heart could be distinguished.

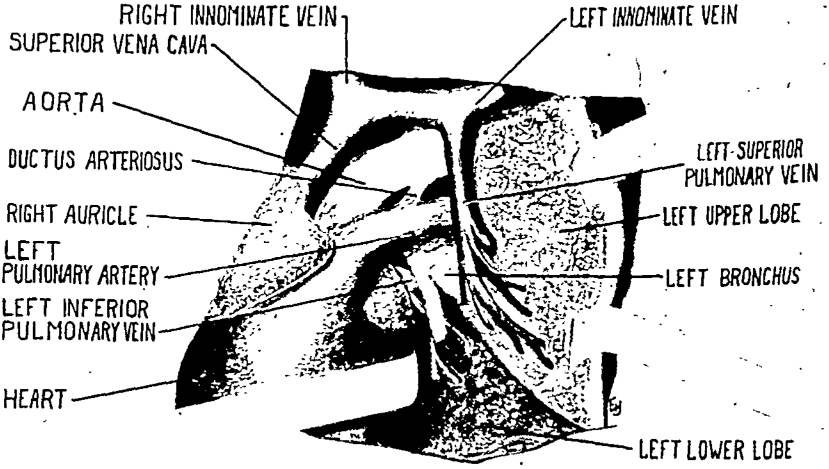

Fig. 3.—Anomalous vessel found in a dissected specimen. Left superior pulmonary vein drains into the left innominate vein. The left inferior pulmonary vein follows its normal path of drainage into the left atrium. (Reproduced from J. THORACIC SURG. 19: 864-874, 1950.)

CASE 2.—The anomalous vessel in this case was found in a 40-year-old white man. It drained the entire left upper lobe and coursed cephalad anterior to the bronchus, left pulmonary artery, and arch of the aorta. It terminated in the left innominate vein 5 cm. from the point where the latter joined the superior vena cava, and measured 4.8 cm. in circumference (Fig. 4).

The left inferior pulmonary vein was present and drained into the left atrium. The ductus arteriosus and foramen ovale were both completely sealed off.

CASE 3.—The following case of an anomalous drainage was seen in an adult male cadaver. The left upper lobe was drained by a left superior pulmonary vein, but instead of emptying into its normal site this vessel took an aberrant course, ascending into the superior mediastinum. In this part of its course it was joined by two vessels; (a) the superior intercostal vein of the left side, and (b) a vein which coursed anterior to the pulmonary artery and extended caudad into the vestigial fold of Marshall. This latter vein, however, was not continuous below with the oblique vein of the left atrium, and therefore, was not a complete persistence of the left superior vena cava. The three vessels observed, namely the left superior intercostal, the remnant of the left superior vena cava, and the left superior pulmonary vein joined to form a single trunk which entered the left innominate vein (Fig. 5).

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*This case was briefly mentioned by Healey and Gibbon in J. THORACIC SURG. 19: 864, 1950.

## SIGNIFICANCE OF ANOMALOUS PULMONARY VEINS

Abnormalities in the number of pulmonary veins are of little clinical significance. Surgically, like abnormalities elsewhere in the body, one should be cognizant of these variations. When the veins are reduced in number, as in the case with a common pulmonary vein, the operative procedure may be simplified. The presence of multiple venous channels, however, may add further difficulties to an already tedious operative procedure.

Anomalous pulmonary-systemic connections do have important clinical significance at present, and may have even more in the future. When there is a complete drainage of the pulmonary circuit into the systemic system, the clinical picture, according to certain investigators, may be quite typical. The fact that an actual diagnosis of this condition during life has been made in only one instance[20] shows clearly that the anomaly has not been sufficiently emphasized in the past. In the near future, further cases will undoubtedly be detected with the aid of modern diagnostic methods of cardiac catheterization and angiocardiography. By these means, one may detect certain changes which are commonly encountered in this malformation, such as a high oxygen saturation of the blood in the right atrium compared with a decreased saturation of the peripheral blood, an increased right atrial pressure, an increase in the size of the right side of the heart, and a dilatation of the superior vena cava (the latter will occur, of course, only if the anomalous vessel drains into the superior vena cava or its tributaries). Electrocardiographic studies exhibit a right axis deviation. With all of these changes there is usually little or no cyanosis, except when accompanied by other congenital anomalies. Because of the pulmonary

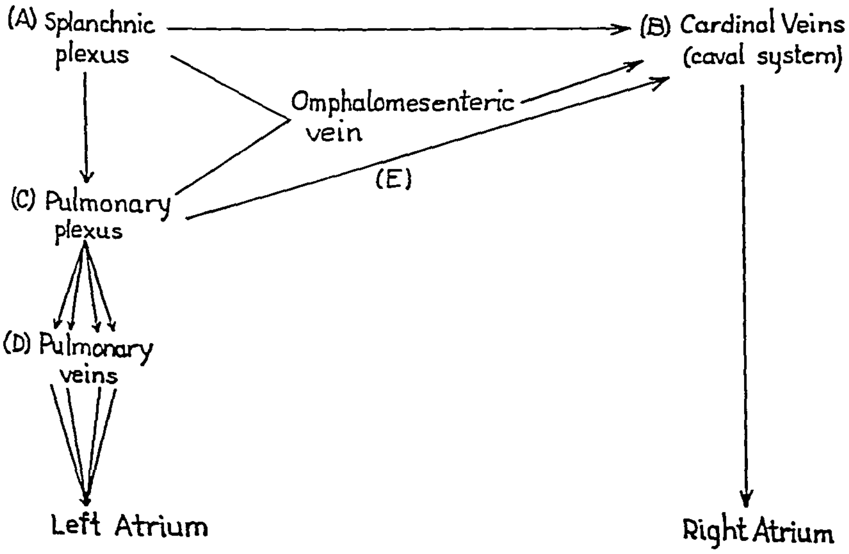

Fig. 6.—Diagrammatic representation of the connections of the pulmonary venous system in the embryo. (See text for explanation.)

The embryology underlying the anomalous connections between the pulmonic and systemic venous systems has been discussed by many investigators. Brown,[6] in a study of the development of the pulmonary veins, described a plexus of capillaries surrounding the intestinal tract of the embryo which he termed the "splanchnic plexus" (Fig. 6,*A*). The cephalic portion of this capillary network drains into the anterior cardinal veins; the caudal portion drains into the posterior cardinal veins and the omphalomesenteric veins (Fig. 6,*B*). These latter veins are destined to become the unpaired portal vein.

With the development of a phylogenetically newer structure—the lung bud—a secondary plexus develops as an extension from the cephalic portion of the splanchnic plexus. This pulmonary plexus (Fig. 6,*C*) will have connections, therefore, with the splanchnic plexus, the cardinal veins, and the omphalomesenteric veins. The capillaries of the pulmonary plexus coalesce to form a longitudinal vein lying at the ventral pole of the pulmonary anlage. This vein passes through the dorsal mesocardium to open into the sinus venosus cephalad to and between the ducts of Cuvier. This common pulmonary vein will give rise to the four pulmonary veins found in the adult which enter the left atrium (Fig. 6,*D*). Most connections between the primary or splanchnic plexus and the secondary or pulmonary plexus are usually obliterated; as well as the connections between the latter and the cardinal and omphalomesenteric veins. Theoretically, any of these vascular channels may persist after birth.

Pertinent here are the observations of Zuckerkandl[23] made in 1881. Investigating the relationship of the pulmonary with the systemic venous system by injecting the pulmonary veins with a colored mass, he noted that the colored mass filled not only the pulmonary veins, but also the bronchial veins and through these the azygos, hemiazygos, and the mediastinal network of veins, all of which are derivatives of the cardinal veins. He claimed, therefore, that certain of the connections of the pulmonary plexus in the embryo, as described previously, may normally persist in the adult. The extent of these anastomotic channels in the adult varies with the individual; but regularly the pulmonary veins anastomose with the mediastinal plexus. The anomalous pulmonary-systemic connections reported in this paper may accordingly be regarded as an overgrowth of one of the normal connections as described by Zuckerkandl (Fig. 6,*E*), with a corresponding underdevelopment in the pulmonary plexus where the pulmonary vein in question should normally develop.

In many instances the pulmonary veins are normal in position at the hilus of the lung, yet enter the right side of the heart instead of the left. This may readily be explained on an embryologic basis. As previously mentioned, the original single pulmonary veins drains into the sinus venosus. With the displacement of the sinus to the right, the pulmonary orifice empties into the left portion of the primitive atrium just to the left of the left sinus valve. The formation of the interatrial septum definitely assigns the opening of the pulmonary vein to the left atrium. In an abnormal developmental procedure, the pulmonary orifice may be shifted to the right and thus account for many of the cases of aberrant drainage of the pulmonary veins into the coronary sinus, right atrium, superior vena cava, and inferior vena cava.

## SIGNIFICANCE OF ANOMALOUS PULMONARY VEINS

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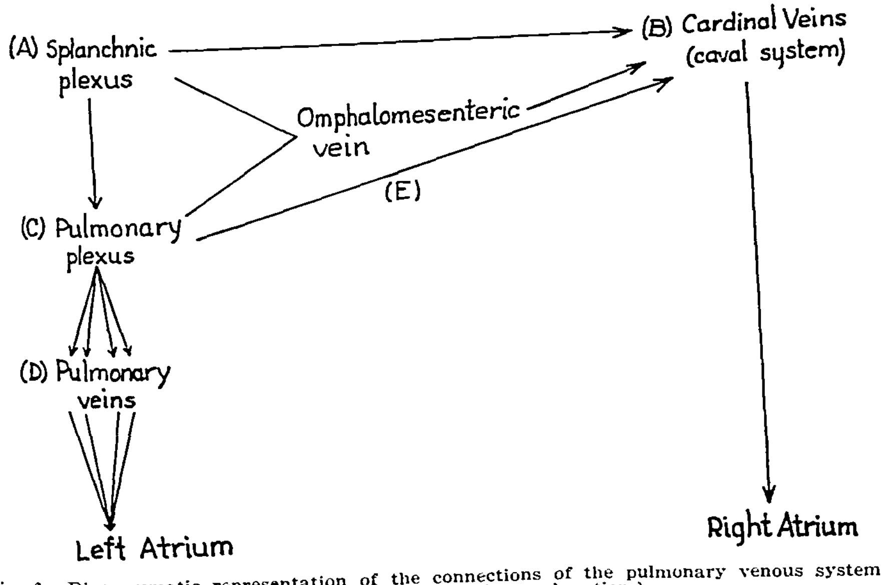

Fig. 6.—Diagrammatic representation of the connections of the pulmonary venous system in the embryo. (See text for explanation.)

congestion which frequently occurs due to increased pulmonary circulation, these cases are often interpreted as pulmonary disease rather than cardiac in origin. The recirculation also places extra strain upon the right side of the heart leading eventually to a right-sided heart failure.

As previously stated, the average age of the majority of these cases was 5.8 months. An operative correction of this deformity to prolong the life of children afflicted with it would certainly be a beneficial one, but thus far such a procedure has not been devised.

In cases where there is an incomplete drainage of the pulmonary system into the systemic system, the clinical signs are by no means typical. Indeed, a case may be asymptomatic depending upon the percentage of pulmonary tissue thus drained. Hughes and Rumore[14] claim that if less than 50 per cent of the pulmonary circuit drains into the right side of the heart, life to adulthood is possible. Those cases of incomplete drainage which are symptomatic may also be corrected surgically. There is only one report in the literature[7] which was reviewed by us, in which there was an attempted surgical correction of this malformation, and this was by ligation of the anomalous vessel. This attempt, however, was not successful. Brantigan[4] suggests either removal of that portion of the lung drained by the anomalous vessel, thereby preventing a right sided heart failure, or better, the malformation may be corrected by anastomosing the anomalous vessel to the left atrium either directly or indirectly.

Aside from the fact that incomplete drainage of the pulmonary into the systemic venous system may give rise to clinical signs and symptoms, it has importance in other ways. Many investigators have emphasized the high incidence of pulmonray disease in lungs which possess certain congenital anomalies. Conant and associates[8] described a case in which a patient had tuberculosis of the left lung. This lung was drained by a common vein which emptied into the left innominate vein. The functional value of this lung was reduced not only by the disease present, but, in addition, by the anomalous drainage of this lung into the systemic system which also increased the work load upon the right side of the heart. When spread of the disease process to the right lung occurred, death shortly ensued.

Such anomalous connections have surgical importance as well. For example, failure to recognize the drainage of a superior pulmonary vein into the systemic venous system might prove fatal when both lower lobes are removed for bronchiectasis.

Within the past two years, sixteen cases have been reported by nine different authors[2, 7, 9, 11, 12, 15, 17, 18, 21] in which anomalous pulmonary veins were diagnosed during life by means of cardiac catheterization, angiocardiography, and roentgenographic methods. All of these cases exhibited an incomplete drainage of the pulmonary circuit. It still remains for us, however, to diagnose those more important cases in infants having complete drainage of the pulmonary veins into the right side of the heart. Nevertheless, these recently reported cases illustrate the fact that anomalous pulmonary veins can be diagnosed in living individuals, and offer assurance that they will be diagnosed more frequently in the future than they have been in the past.

## SUMMARY

1. A general anatomic survey of anomalous pulmonary veins is presented; cases previously reported in the literature have been classified, and their incidence cited.

2. Three cases of anomalous-pulmonary-systemic connections observed by the author are described.

3. The clinical and surgical significance of anomalous pulmonary veins are briefly discussed.

## ADDENDUM

Since submitting this paper, we have observed one case exhibiting a complete anomalous drainage of the pulmonary veins into the right side of the heart, and have collected four additional cases from the literature. In regard to the incomplete type of anomalous drainage, we have seen two cases and found two others reported within the past year. In addition, twelve cases have been recorded which were diagnosed during life by means of cardiac catheterization and/or angiocardiography.

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# BRONCHOGRAPHY IN BRONCHIAL CANCER AND COLLATERAL VENTILATION

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The following three case histories are of practical and theoretical importance.

Case 1.—A man, aged 50 years, had been troubled with a bad cough five months previous to examination. His complaints completely disappeared, but three months previously he had felt a slight pain in the right side of his chest. He did not expectorate any blood. He felt tired, had little appetite, and lost 3 kilograms in weight in a few months' time. For this reason his physician sent him to the tuberculosis center. On examination, percussion and auscultation of the chest revealed no anomalies. A roentgenogram taken at this time showed an obvious anomaly, viz., an irregular shadow around the right hilus.

From the planigrams of the chest it appeared that the shadow around the hilus consisted of a conglomeration of globular shadows. On a section 16 cm. from the back, the impression was that the anterior side branch of the upper-lobe bronchus on the right, which lay within this mass of shadow, was completely occluded.

For bronchography the right side only was filled with 10 c.c. of lipiodol. The three usual photographs were taken, the dorsoventral one, the lateral one, and the oblique one. On all three photographs a stop was visible in the anterior side branch of the upper-lobe bronchus. Fig. 1 shows the dorsoventral photograph. The shadow around the hilus immediately strikes the eye. The right upper-lobe bronchus looks slightly compressed. The stop in the anterior side branch (*3*) is indicated by an arrow. Fig. 2 is the lateral photograph. On this one, too, the shadow is clearly visible. The anterior side branch is completely absent on this bronchogram, whereas all other side branches are well filled. Further toward the periphery in the anterior segment of the upper lobe not a sign of a shadow is visible.

When bronchoscopy was performed the right upper-lobe bronchus was immediately aimed at. The spur of the upper-lobe bronchus seemed slightly thickened. With the telescope the bifurcations were visible. The entrance of the anterior side branch was considerably swollen. The secretion was removed by suction for bacteriologic and cytologic examination (Keuning's opinion was: suspected carcinoma). Next the upper-lobe bronchus was focused at its maximum. With the curette, some tissue from the anterior side branch was removed. The microscopic diagnosis (Vos) was: carcinoma solidum. After complete functional examination in the internal clinic (Orie), pneumonectomy was performed by Eerland with favorable results to the present time. During the operation many carcinomatous glands were found near the hilus, which were also removed as far as visible and palpable.

A few points from this case history deserve closer consideration. A striking feature is the vague anamnesis. The complaints of pain in the chest were the most preponderant ones. This is a symptom that we have also often seen in cases of bronchial carcinoma. In this patient it was probably due to irritation of the pleura.

From a bronchologic point of view, two points are of special importance. This patient is a typical example of the success of what we nowadays call:

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*directed bronchoscopy.* On the grounds of a localization, accurately determined by x-ray examination (planigraphy and bronchography) a certain bronchus is directly aimed at, and here some tissue is blindly removed by means of a curette. This directed bronchoscopy, as also indicated by Soulas and Versteegh from Bronkhorst's Utrecht school, is an important addition to the diagnostics. In a large number of cases of bronchial carcinoma the bronchoscopy is very simple. The tumor is immediately visible, and biopsy can easily be performed. Difficulties are chiefly met with when the localization is in the upper lobe and more toward the periphery in the middle and lower lobes. In these cases one can reach further in this way. As a rule we perform this blind biopsy with a small curette. Its importance is sufficiently clear from the results obtained from a continuous series of 160 patients with bronchial carcinoma seen in the clinic during the last few years.

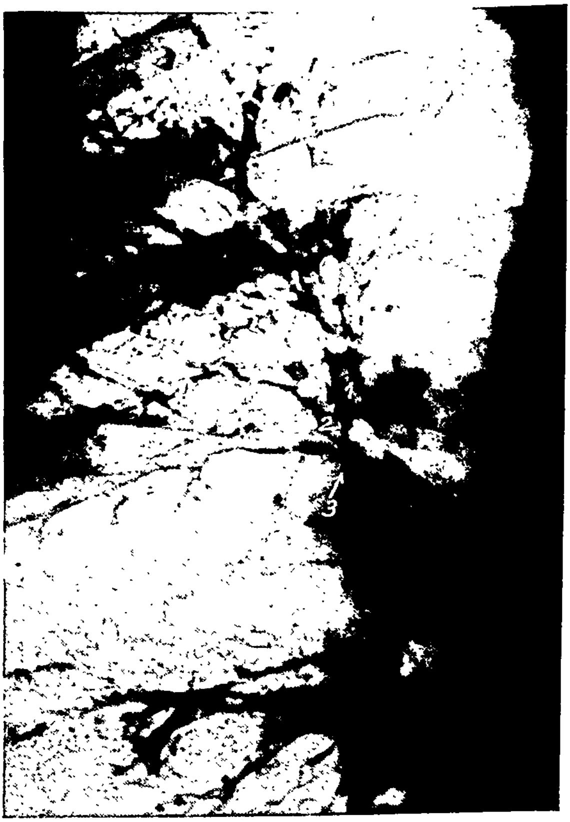

Fig. 1.—Case 1. Dorsoventral bronchogram, right side.

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|---|---|
| Total number of patients | 160 |
| Bronchography | 110 |
| Bronchoscopy | 154 |
| Microscopy + | 123 = 80 per cent |
| Biopsy + | 106 |
| Curettage + | 17 |

As appears from these statistics, bronchoscopy was performed on nearly all patients. In six cases the diagnosis could also be established with certainty without it, e.g., by means of an excision from supraclavicular glands. On most patients bronchography was performed. This is chiefly done for two reasons. First with an eye to the "directed bronchoscopy." For this reason bronchography is usually performed before bronchoscopy. Second, the bronchogram often shows various anomalies, that are sometimes of great importance, e.g., in determining the operability. The second case clearly demonstrates this fact.

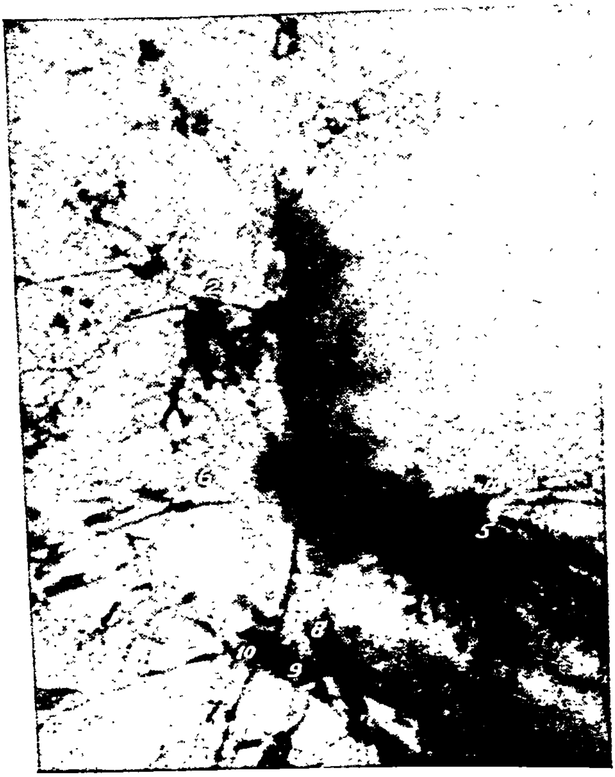

Fig. 2.—Case 1. Lateral bronchogram.

The result that in 80 per cent of these cases it was possible to establish a certain diagnosis by means of bronchoscopy is very satisfactory. This high percentage was also found of late years at Utrecht by Versteegh and Swierenga and, among others, by Holinger and Soulas. This is only due to the improved technique of the last few years. Until recently the usual percentages given were 50 to 60 per cent. It appears that an important addition is furnished by the blind biopsy, 17 times on 123 patients. It seems fairly certain that this percentage will not increase in the future, on the contrary, it will decrease. Lately we see more and more difficult cases with tumors in the periphery. This is chiefly a result of the ever-increasing group fluoroscopy of the population of

Holland. Fortunately, we now have cytology at our disposal, which was introduced into bronchology by Clerf and Herbut. The Groningen results are in the main very good, thanks to the cooperation with the histologist Keuning. In these cases it is also important to perform directed bronchoscopy in order to remove the secretion from the suspect bronchus.

In a quite different respect this case is also important. The shadow, visible in the right half of the chest, is completely caused by the tumor and the metastases in the hilar glands. There is no shadow as a result of anomalies in the pulmonary tissue of the anterior segment of the upper lobe. This fact is remarkable. A big side branch completely, or nearly so, occluded has still not resulted in atelectasis. In these cases the possibility should always be taken into account that ventilation is still possible alongside the obstruction. This fact was called attention to by my friend Bronkhorst by a demonstration of two evident cases. Personally, I have always seen atelectasis in cases of a complete stop of a main or lobar bronchus. Atelectasis was often seen even when lipiodol did still pass through the stenosis. So this is just the reverse. It is more plausible to explain the fact that an occluded part of the lung contains air, by ventilation by way

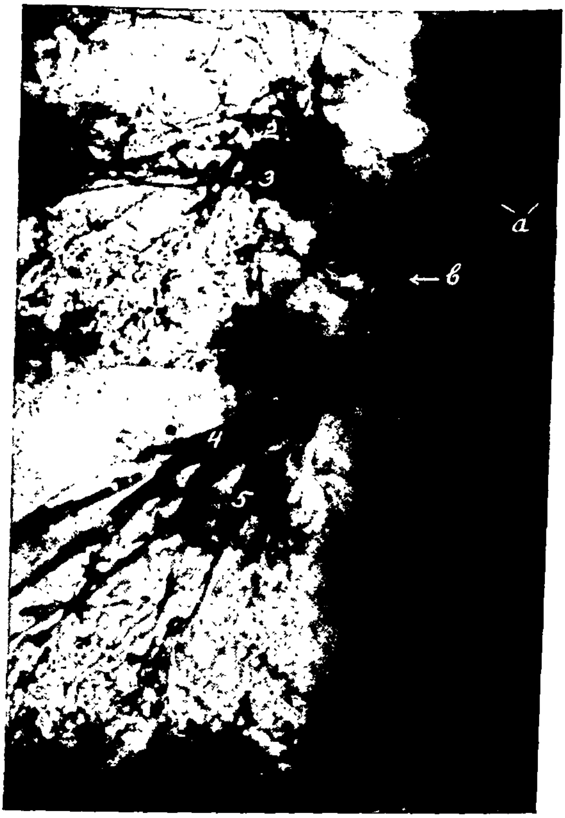

Fig. 3.—Case 2. Dorsoventral bronchogram, right side.

of the pulmonary tissue from out of the other segments of the upper lobe. This is collateral ventilation, to which particular attention has been paid in Groningen, thanks to the cooperation of Dirken.[1] It was first described by van Allen and his collaborators.[2]

CASE 2.—A man, aged 59 years, had had his complaints for a short time only that he had had a bad cough. The internist found no anomalies when examining the chest, but on the roentgenograms he found a broadened hilus, particularly on the right. Fig. 3 also clearly shows this shadow. But there is no shadow in the lower part of the lower lobe, the space between heart and diaphragm is occupied by air-containing tissue. Fig. 3 shows the dorsoventral bronchogram. This reveals a number of anomalies. In the first place the carina is considerably broadened (*a*). The right upper-lobe bronchus is well filled, the three segmental bronchi show no anomalies. Somewhat lower down the stem bronchus suddenly grows much narrower with a quite smooth margin, apparently the bronchus is compressed from the medial side (*b*). On the place of the bifurcation of the middle- and lower-lobe bronchi medially a small irregularity is visible. One has some difficulty in further finding one's way in the bunch of downgoing bronchi. The lateral (*4*) and the medial (*5*) branches of the middle lobe bronchus are still recognizable with fairly great probability. Behind these, however, there are two more descending bronchi that cannot be explained as basal branches. The lateral photograph gives the solution. Above, one sees the main bronchus with the entrance of the upper-lobe bronchus. The basal bronchi are completely absent, in the lower-lobe

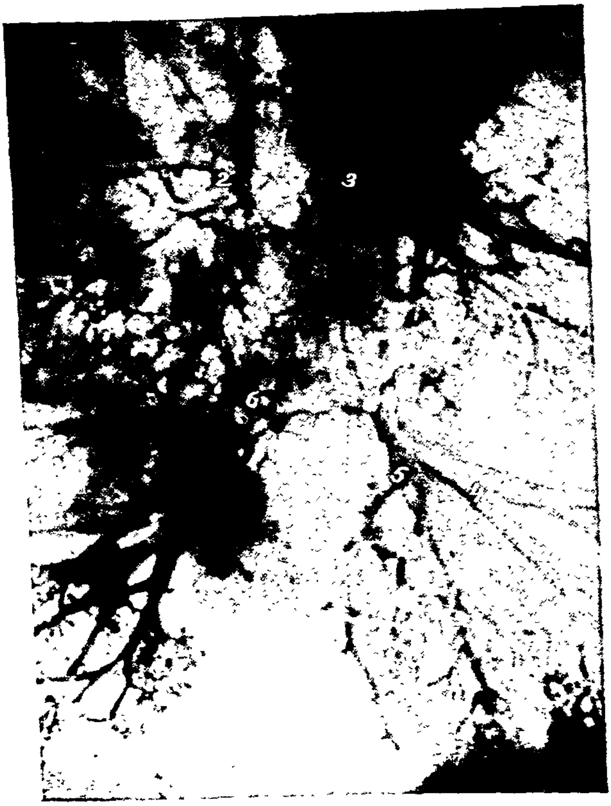

Fig. 4.—Case 2. Lateral bronchogram.

bronchus there is an obstruction with an irregular margin. Only the middle-lobe bronchi (*4* and *5*) and the apical branch of the lower lobe (*6*) are filled. The entrances of both bronchi, but particularly that of the middle lobe, are slightly narrowed. The two descending branches on the dorsoventral bronchogram of Fig. 3 are, therefore, side branches of the apical bronchus of the lower lobe. The occluded part of the lung contains the four basal segments. This part of the lung is smaller than normal, for the middle lobe covers the anterior part of the diaphragm. Apparently there is hypoventilation in the occluded part of the lung, but, thanks to the collateral ventilation, it still contains air. Owing to this the physical and x-ray examinations of the chest revealed no phenomena of occlusion of the lower-lobe bronchus.

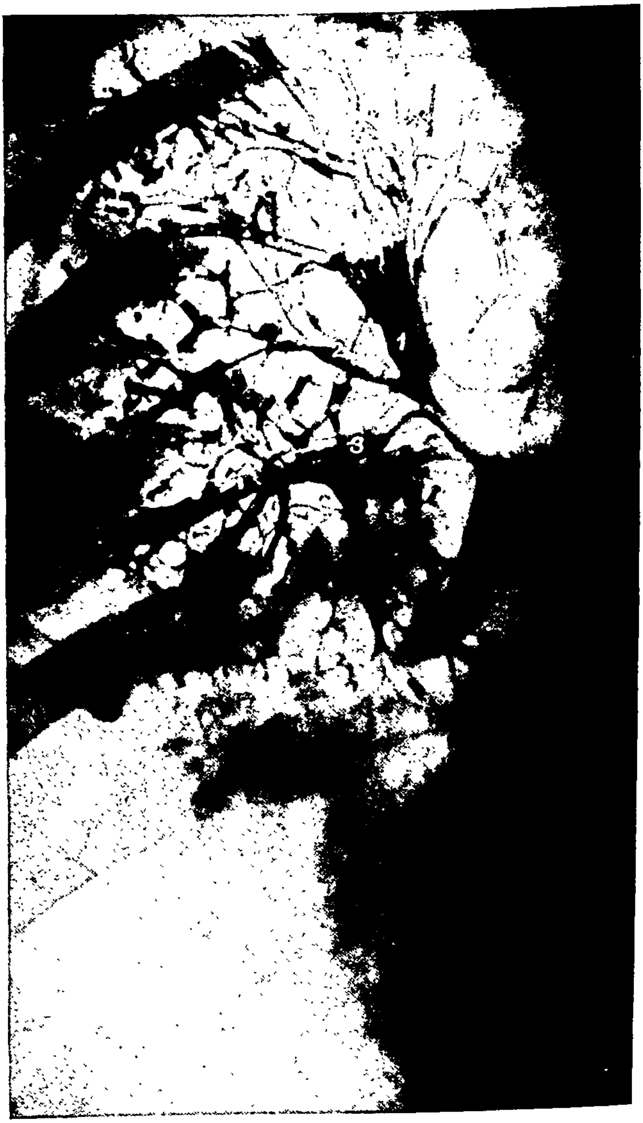

Fig. 5.—Case 3. Dorsoventral bronchogram, right side.

Bronchoscopy revealed no anomalies of the trachea. The carina was considerably broadened. The stem bronchus was narrowed. Immediately below the bifurcation of the middle-lobe bronchus and the apical branch of the right lower lobe the bronchus was completely occluded by easily bleeding granulations. Biopsy revealed a planocellular carcinoma. Owing to the bronchologic examination it was, therefore, possible not only to establish the diagnosis with certainty, but it was also apparent that the patient could not be operated upon owing to the extension into the mediastinal glands (broadened carina and pressure on the stem bronchus).

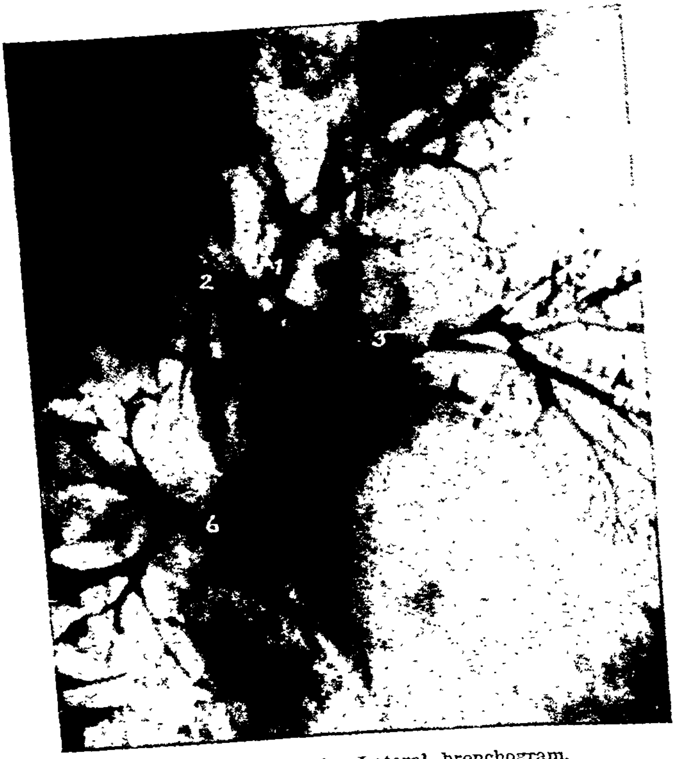

Fig 6.—Case 3. Lateral bronchogram.

Case 3.—A 52-year-old man had a productive cough for a few months, with no hemoptyses. On the right side of the heart a perihilar shadow was visible. This shadow, which is very suspect for atelectasis of the middle lobe, is clearly visible on the bronchogram of Fig. 5. Fig. 5 is a dorsoventral photograph, showing that the right lower lobe contains a fair quantity of air. The space between heart and diaphragm is not portrayed. In this photograph the obstruction is clearly visible; it has a conical shape. The impression is conveyed that an occlusion exists in the stem bronchus (with this term is meant, in Groningen, the main bronchus of the right side below the bifurcation of the upper-lobe bronchus, so the common bronchus of the middle and lower lobes). Fig. 6 is the lateral photograph, demonstrating once more, just as in the preceding case, the importance of taking photographs in various directions, also in bronchography. The picture thus obtained is quite different. There is not one obstruction, but two: an occlusion of the middle-lobe bronchus and an occlusion of the lower lobe bronchus below the apical branch (6). With bronchoscopy a tumor, occluding the lower-lobe bronchus below the branching off of the apical branch, was found. Biopsy revealed a carcinoma.

This case is particularly demonstrative, as it so clearly shows the difference between the occlusion of the middle-lobe bronchus, in which case collateral ventilation is impossible, resulting in atelectasis, and the occlusion of the lower-lobe bronchus, in which case collateral ventilation is still possible through the apical branch (*6*), thus probably prohibiting atelectasis. From the surgical preparation it was stated that the occlusion in the lower lobe was caused by the primary tumor and the occlusion in the middle lobe by glandular metastases.

Naturally these cases are exceptions in the great number of patients suffering from bronchial carcinoma now seen in the clinic. They are, however, of great theoretical importance. An important point is that very considerable bronchial stenosis may exist, whereas an examination, also with roentgenograms, does not reveal any signs of bronchial stenosis. This is in all probability caused by collateral ventilation. This condition can only have a temporary existence. For, as was already described by van Allen, an inflammation easily eliminates the collateral ventilation. Bronchial stenosis is nearly always accompanied by inflammation. Further growth of the tumor, moreover, will after some time obturate the apical branch from the lower lobe thus certainly eliminating collateral ventilation.

In the preceding communication,[1] the importance of collateral ventilation in cases of foreign body was pointed out. We saw several examples of it. The above cases make it clear that collateral ventilation is also important in cases of tumors.

## SUMMARY

Three cases of bronchial carcinoma are described, in which probably collateral ventilation existed. In cases of bronchial carcinoma bronchoscopy is as a rule preceded by bronchography. Bronchography can often furnish important indication, also in cases of carcinoma, e.g., concerning the operability. It is of particular importance with tumor in the periphery for the performance of a directed bronchoscopy. Its importance is demonstrated by the statistics of our last 160 cases in which, thanks to this directed bronchoscopy, a positive biopsy was possible in 80 per cent.

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# PHYSIOLOGIC STUDIES FOLLOWING THORACIC SURGERY

## I. Immediate Effects of Thoracoplasty

William W. Stead, M.D., and Philip H. Soucheray, M.D.
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### INTRODUCTION

THIS is the first of a series of reports dealing with the physiologic effects of thoracic surgical procedures employed in the treatment of cavitary pulmonary tuberculosis. The effects produced by lobectomy, pneumonectomy, and segmental resections will be covered in subsequent communications. In the choice of a surgical procedure suitable for a given patient three factors must be considered: (1) The nature of the tuberculous lesion and the merits of the various surgical approaches as definitive treatment for the lesion at hand[1]; (2) the pulmonary reserve of the patient and his ability to withstand the permanent loss of function following the operation; and (3) the magnitude of the immediate stress which attends the procedure, and the ability of the patient to withstand this stress. With these considerations, the procedure is then designed to fit both the lesion itself and the patient as a whole. It was felt that a careful study of the physiologic effects of the several surgical approaches to the treatment of cavitary pulmonary tuberculosis would, therefore, be of value.

Excisional surgery is rapidly gaining in popularity in the treatment of tuberculosis. Much is being written upon the specific indications for resection and upon its efficacy in the treatment of the various types of tuberculous lesions. In the presence of normal pulmonary reserve, most surgical procedures present little difficulty, but in many cases diffuse pulmonary disease (emphysema and fibrosis) coexists with the tuberculous lesion under attack. It is this coexisting disease that frequently impairs the over-all pulmonary function and is, therefore, the determining factor in the patient's ability to withstand major thoracic surgery.[1, 2] A great deal of experience is needed in assessing the ability of a borderline case to withstand the resection of pulmonary tissue. Thus, in selecting the ideal operation for the treatment of a given tuberculous lesion, a compromise must frequently be made in order to keep the immediate surgical trauma and ultimate loss of pulmonary function within the limits imposed by the patient's over-all pulmonary reserve. The purpose of this series of reports has been to quantitate and compare the immediate stress and the ultimate sacrifice of pulmonary function produced by the various operations currently in use in the treatment of cavitary pulmonary tuberculosis.

The present report is concerned with the physiologic effects that were noted within twenty-four hours of each stage of the three-stage thoracoplasty.

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Thoracoplasty was chosen for the first study because it is a well standardized and familiar procedure in thoracic surgery. The magnitude of the stress imposed by thoracoplasty is so well known to chest surgeons that it may serve as a yardstick for the quantitative comparison of the effects of thoracic operations of greater magnitude.

The observations herein reported may be divided into two parts: (1) The preoperative determination of pulmonary function, and (2) the evaluation of the physiologic status of the patient sixteen to twenty hours postoperatively. The preoperative study served two purposes: (a) to aid in the objective estimation of the pulmonary reserve, and (b) to furnish a base line for immediate postoperative observations and for subsequent analysis of the ultimate functional changes to be reported at a later date.

## METHODS

A few days prior to the first stage of thoracoplasty, the patient was taken to the laboratory at 7:30 A.M. in the fasting state. Weight, height, age, and vital capacity were recorded. The functional residual volume and index of pulmonary mixing were determined in duplicate in the manner described by Cournand and associates.[3] The subdivisions of the vital capacity were measured from the tracing made on a standard Collins or McKesson metabolimeter. This first group of tests served to acquaint the subject with the respiratory apparatus, in order that he might be more relaxed during subsequent observations.

The following morning the patient was brought to the laboratory in the fasting state. The oral temperature was recorded. A large bore needle (Cournand modification of the Linde needle) was inserted into the femoral artery under local anesthesia. This needle was left in place throughout the entire procedure.

The expired air was then collected for a seven-minute period in the Tissot gasometer, which had previously been thoroughly rinsed with the patient's expired air. An open circuit was used, the patient inhaling room air through a valve of 53 c.c. dead space. During this period the pulse and respiratory rates were recorded, and two samples of arterial blood were withdrawn through the Cournand needle without disturbing the patient. Resting ventilation and basal metabolic rate were determined by measurement and quantitative analysis of the expired gas collected in the Tissot gasometer. The physiologic dead space and alveolar ventilation were also calculated from these data (see below).

Next, the subject inhaled 100 per cent oxygen through a similar open circuit. After a period of at least seven minutes, a third sample of arterial blood was withdrawn, and the needle was removed. This sample was analyzed at once for oxygen content to determine the amount of oxygen physically dissolved in the blood. This value was used in the calculation of the pulmonary shunt, after the method of Fasciolo and Chiodi,[4] using the formula:

$$X = \frac{V_{aO_2} - V_{vO_2}}{V_{Pa_{O_2}} - V_{vO_2}} \times 100$$ where: X is per cent of blood passing through aerated lung tissue;

$V_{aO_2}$ is arterial blood $O_2$ content of patient; $V_{vO_2}$ is venous blood $O_2$ content (actually in this study $V_{aO_2} - V_{vO_2}$ was not determined, but a value of 4 volumes per cent was assumed instead); $V_{Pa_{O_2}}$ is arterial hemoglobin capacity plus the mean value for dissolved $O_2$ found in a series of normal subjects. This is taken as the theoretical normal for this patient if he had only a normal amount of pulmonary shunt.

With the patient in a sitting position, the maximum breathing capacity was measured directly from the volume of air collected in a Douglas bag during thirty seconds of maximal voluntary hyperventilation. The resistance in the valve system was minimal at the rate of ventilation achieved by most of our subjects.

The blood for the determination of the pH was drawn into a 10 c.c. syringe which had been warmed to 37° C. It was agitated briefly with a small amount of mercury and 0.1 c.c. heparin, and the pH determination carried out within five minutes at 37° C. in the manner described by Wilson and Ognanovich.[5] In a series of normal subjects the mean pH by this method was 7.40 ± 0.02.

The arterial blood carbon dioxide content was determined immediately in duplicate after the method of Van Slyke and Sendroy.[6] The oxygen content and hemoglobin capacity were determined in duplicate by the method of Van Slyke and Neill.[7] Originally a correction was made for change in hemoglobin concentration which occurs in the tonometer in the manner described by Comroe and Walker[8] and a 2 per cent higher saturation found by this method. This was felt to contribute nothing, and so values are expressed in the conventional manner. The normal value in this laboratory is 96.50 per cent uncorrected and 98.50 per cent corrected for the change in hemoglobin concentration.

The oxygen content and capacity of the blood collected during the breathing of pure oxygen were analyzed. The oxygen content of the blood collected during the breathing of 100 per cent oxygen, minus the hemoglobin capacity, was taken as an estimate of the oxygen which was physically dissolved in the blood. The normal value for the dissolved oxygen under these conditions is 1.80 ± 0.294 volumes per cent in this laboratory. The true value should be 2.04 volumes per cent under these circumstances if all factors are taken into account.[9] This method, however, does give a value which is fairly constant in normals and in the patients preoperatively. The fact that little blood passes through the diseased portion of the lung has been observed by Berggren[10] and also by Björk.[11]

In determining the maximum breathing capacity, the thirty-second period was chosen because it would give sufficient gas for accurate measurement and would at the same time avoid gross fatigue on the part of the patient. The normal values in this laboratory are somewhat lower than those given by Cournand and Richards.[12] This is probably because the method used by these investigators involved the rebreathing of expired air in a spirometer, and the method employed here utilized fresh room air for each breath. In this manner, the additional stimulus of increased carbon dioxide and decreased oxygen content of the inspired gas was avoided. Parallel determinations in this laboratory using both methods showed that the spirometric method of Cournand gave consistently higher values.

Arterial blood $P_{CO_2}$ ($Pa_{CO_2}$) was estimated from the values for arterial blood $CO_2$ content and pH. The normogram prepared by Singer and Hastings[13] was used for convenience. Alveolar $P_{CO_2}$ ($P_{A_{CO_2}}$) was assumed to be equal to arterial blood $P_{CO_2}$ after the suggestion of Riley.[14] $CO_2$ excretion per minute ($\dot{V}_{CO_2}$) was determined directly by measurement and analysis of the expired air. Alveolar ventilation per minute was calculated by use of the formula:[14]

$$\dot{V}_A = \frac{\dot{V}_{CO_2}}{F_{A_{CO_2}}}$$

where: $\dot{V}_A$ = alveolar ventilation in cubic centimeters per minute.
$\dot{V}_{CO_2}$ is rate of $CO_2$ excretion in cubic centimeter per minute.
$F_{A_{CO_2}}$ is fraction of alveolar gas made up by carbon dioxide.
$\dot{V}$ is resting minute volume of ventilation in cubic centimeters per minute at standard condition of temperature and pressure.

Alveolar ventilation per breath was calculated by use of the formula:

$$V_A = \frac{\dot{V}_A}{f}$$

where: $V_A$ is alveolar ventilation per breath.
$f$ is respiratory rate per minute.

TABLE I

| | MEAN NORMAL | MEAN PREOPERATIVE | MEAN POSTOPERATIVE NO. 1 | MEAN POSTOPERATIVE NO. 2 | MEAN POSTOPERATIVE NO. 3 |
|---|---|---|---|---|---|
| Oral Temp. (°F.) | | 98.3 ± .4 | 100.1 ± .8* | 99.2 ± .7* | 99.6 ± .7* |
| Pulse Rate/Min. | | 81. ± 10.2 | 108. ± 13.* | 108. ± 10.* | 113. ± 12.* |
| Metab. Rate $\frac{(\text{Cal/hr.})}{M^2}$ | 35.0 ± 2.7 | 39.2 ± 3.2 | 45.7 ± 2.8* | 44.1 ± 2.6* | 49.3 ± 8.4* |
| Resp. Quotient | .90 ± .04 | .87 ± .08 | .76 ± .03* | .80 ± .06* | .78 ± .08* |
| Arterial Blood: | | | | | |
| pH | 7.40 ± .01 | 7.40 ± .01 | 7.40 ± .01 | 7.40 ± .01 | 7.39 ± .01 |
| $CO_2$ cont. MM/L | 21.3 ± 1.0 | 21.3 ± 1.1 | 21.4 ± .5 | 21.3 ± .9 | 21.5 ± 1.6 |
| $P_{CO_2}$ (mm. Hg) | 39.0 ± 2.0 | 40.6 ± 2.5 | 41.7 ± 3.0 | 40.6 ± 2.0 | 41.4 ± 4.1 |
| Hgb sat'n (%) | 96.5 ± .8 | 95.7 ± 1.7 | 90.3 ± 2.1* | 91.1 ± 1.5* | 90.7 ± 2.9* |
| Pulm. Shunt (%) | 0 | 0.5% | 14.4%* | 17.0%* | 14.5%* |
| $\dot{V}$ (c.c./min.) | 5641. ± 601. | 6180. ± 892. | 7058. ± 856.* | 7543. ± 1190.* | 7147. ± 1302.* |
| f (Resp./min.) | 12. ± 1.8 | 14.5 ± 1.7 | 18.9 ± 3.8* | 18.8 ± 2.6* | 21.0 ± 9.4* |
| $V_T$ (c.c.) | 499. ± 58. | 430. ± 83. | 379. ± 66. | 407. ± 58. | 384. ± 103. |
| $V_D$ (c.c.) | 144. ± 32. | 127. ± 49. | 125. ± 36. | 144. ± 47. | 135. ± 33. |
| $\dot{V}_D$ (c.c./min.) | 1605. ± 421. | 1828. ± 700. | 2355. ± 733.* | 2719. ± 919.* | 2786. ± 619.* |
| $V_A$ (c.c.) | 310. ± 47. | 257. ± 59. | 207. ± 36.* | 211. ± 27.* | 203. ± 63.* |
| $\dot{V}_{CO_2}$ (c.c./min.) | 212. ± 23. | 217. ± 37. | 226. ± 24. | 227. ± 33. | 237. ± 67. |
| $\dot{V}_A$ (c.c./min.) | 3496. ± 381. | 3685. ± 764. | 3835. ± 500. | 3961. ± 675. | 4204. ± 1448. |
| Vital Capacity c.c./$_{M^2}$ | 2290. ± 274. | 1820. ± 360.† | | | |
| Resid. Volume c.c./$_{M^2}$ | 680. ± 148. | 1070. ± 480.† | | | |
| Reserve Volume c.c./$_{M^2}$ | 462. ± 116. | 355. ± 150. | | | |
| Total Lung Vol. c.c./$_{M^2}$ | 2970. ± 313. | 2950. ± 570. | | | |
| $RV/_{TLV}$ % | 23.0 ± 4.1 | 35.9 ± 12.4† | | | |
| Index Pulm. Mixing (% $N_2$) | 1.45 ± .23 | 2.83 ± .88† | | | |
| Max. Br. Cap. L/min./$_{M^2}$ | 61. ± 6. | 41. ± 12.† | | | |

*Statistical analysis reveals this mean to differ significantly from the preoperative mean ($p = 0.05$ or less).

†Statistical analysis reveals this mean to differ significantly from the mean value obtained in sixteen normal subjects ($p = 0.05$ or less).

Physiologic dead space was calculated by use of the formula:

$$V_D = V_T - (V_A - DS_I)$$

where: $V_D$ is physiologic dead space in cubic centimeters.
$V_T$ is tidal volume
$V_A$ is alveolar ventilation per breath
$DS_I$ is dead space of respiratory valve (53 c.c. which when corrected to BTPS is 46 c.c.)

The dead space ventilated per minute was calculated by use of the formula:

$$\dot{V}_D = V_D \times f$$

where: $\dot{V}_D$ is cumulative volume of dead space ventilated in cubic centimeters per minute.

These calculations, although apparently complex, yielded the most useful information obtained from this study for understanding the physiologic changes which follow surgical procedures involving the thoracic cage.

The postoperative observations were made in the patient's room at 7:30 A.M., on the morning following surgery. These consisted in the recording of the temperature, pulse, respiratory rate, and vital capacity. The expired air was collected and arterial blood was drawn in the same manner as described above. The oxygen dissolved in the blood during the inhalation of 100 per cent oxygen was again determined. Each patient was studied in the fasting state.

## CLINICAL MATERIAL

Ten male patients, of ages 23 to 62 years, were studied. Each had previously been reviewed and accepted for thoracoplasty by members of the medical and thoracic surgical services. Each patient had predominately unilateral, upper-lobe, cavitary pulmonary tuberculosis. The contralateral lung was either clear or contained apparently stable tuberculous lesions. There was no significant pleural involvement. Clinical observations showed the patients to vary from men who were physiologically normal to men who were dyspneic on slight exertion.

Normal values for this laboratory were determined by testing sixteen normal young males. These figures were used as a standard by which to judge the pulmonary function of the patients before surgery.

Table I gives the mean values for the preoperative tests in this group of patients, and the mean value found in the group of normal subjects.

It can be seen that the index of pulmonary mixing, and residual volume were significantly greater in this group of patients than in the normal subjects. Several of the patients actually had moderately severe pulmonary emphysema with a residual volume greater than 40 per cent of the total lung volume. The mean vital capacity and maximum breathing capacity were significantly lower than in the normals. The resting minute volume of ventilation, basal metabolic rate, and ventilatory equivalent for oxygen did not differ significantly from the normal values. The arterial blood oxygen saturation, carbon dioxide content, carbon dioxide tension, and pH were also within normal limits. The mean for the oxygen dissolved in arterial blood by breathing 100 per cent oxygen was not significantly different from the normal. This indicated that there was no significant pulmonary shunt in these patients.

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†Statistical analysis reveals this mean to differ significantly from the mean value obtained in sixteen normal subjects ($p = 0.05$ or less).

The mean oral morning temperature rose from 98.3° F. preoperatively to 99.8° F. the day following surgery (Fig. 1*A*). This change, though slight, was statistically significant. The mean preoperative pulse rate was 81 per minute as compared with a mean of 109.6 postoperatively (Fig. 1*I*).

The mean carbon dioxide content of arterial blood in the preoperative period was 21.34 milliequivalents per liter and postoperatively was 21.38 milliequivalents per liter (Fig. 1*D*). The pH of the arterial blood remained unchanged at 7.40 postoperatively (Fig. 1*E*) and the carbon dioxide tension rose only from 40.55 mm. Hg preoperatively to 41.24 mm. Hg the morning after surgery (Fig. 1*F*). None of these changes was of significance.

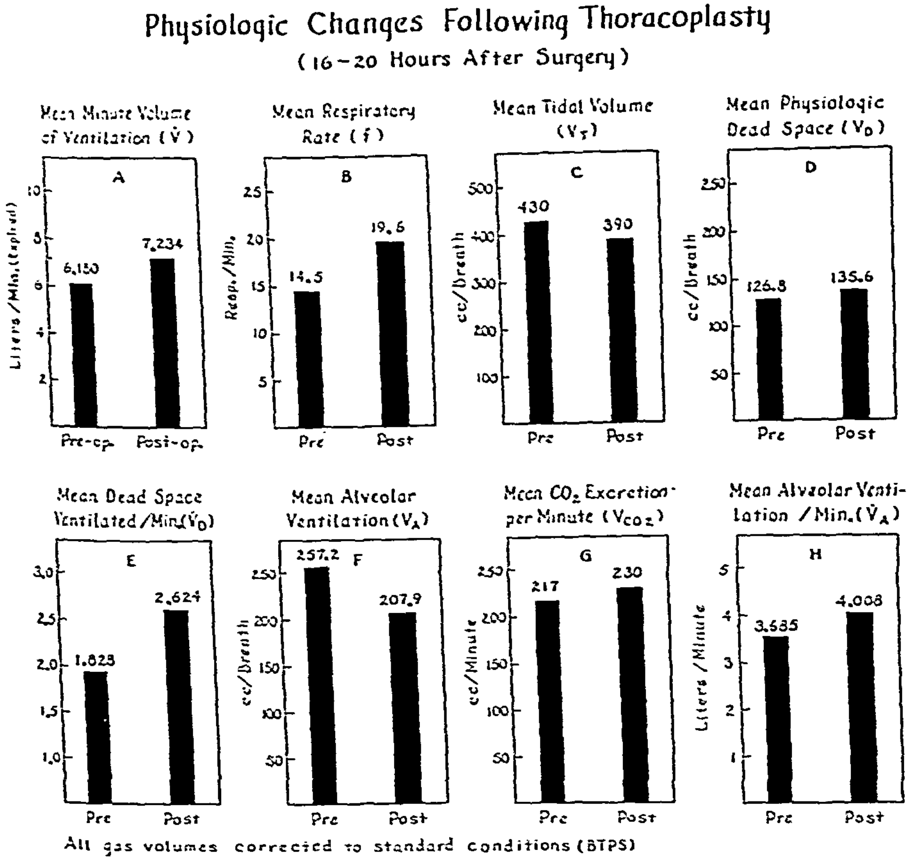

Fig. 2.—Comparison of the ventilatory mechanism of the preoperative and postoperative states. The postoperative figures represent the weighted means of the three postoperative determinations in each subject, making a total of twenty-nine postoperative studies in the ten subjects.

The mean arterial hemoglobin saturation in the first twenty-four hours after the surgical procedure fell to 90.31 per cent from the control mean of 95.07 per cent, while the patient was breathing ambient air (Fig. 1*G*). This change was of significance.

The technique of thoracoplasty used in the patients comprising this study was as follows: The first stage consisted of the removal of the first rib and posterolateral portions of ribs 2, 3, and 4. The second stage was done from twelve to sixteen days later and consisted of the removal of the anterior segments of ribs 2 and 3 and cartilages of 1, 2 and 3. At the third stage, performed twelve to sixteen days following the second, the posterolateral segments of ribs 5, 6, and 7 were removed. It is felt by the Thoracic Surgery Service that these three stages divide the surgical trauma into approximately equal parts. Local infiltration with 1 per cent procaine was the only anesthetic employed.

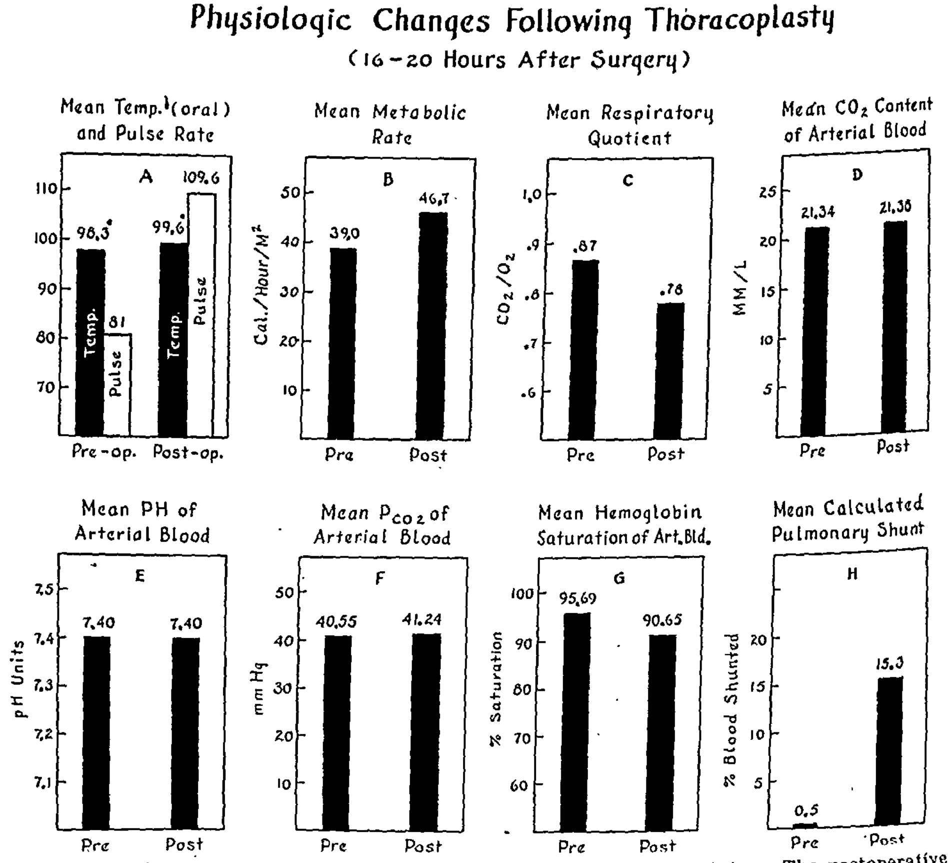

Fig. 1.—Comparison of the preoperative with the postoperative states. The postoperative figures represent the weighted means of the three postoperative determinations in each subject making a total of twenty-nine postoperative studies in the ten subjects. In one subject (M. J.) a pneumonectomy was performed at the time of the third stage thoracoplasty, and the third study is therefore not included in this report.

## RESULTS

The mean physiologic effects of the individual stages are given in Table I. Detailed analysis of the results by stages revealed that there was no difference between the effects produced by the various stages of thoracoplasty. The results of the mean values by stages have, therefore, been averaged and compared with the preoperative control value. These comparisons are given in Figs. 1 and 2.

The foremost function of the lungs is the absorption of oxygen and the elimination of carbon dioxide. Of these, the carbon dioxide excretory factor is of the greater importance since oxygen is easily supplied therapeutically. The lungs are solely responsible for the elimination of carbon dioxide and thus play a vital role in the maintenance of the acid-base balance of the blood. There is as yet no way whereby the excretion of carbon dioxide can be facilitated save by an increased alveolar ventilation. Furthermore, for practical purposes, sole reliance for such ventilation must be placed upon the natural mechanisms of the body. If these mechanisms prove insufficient, carbon dioxide retention and respiratory acidosis ensue.

Two recent papers[16, 17] have called attention to the development of respiratory acidosis *during* intrathoracic operations. The authors felt that the carbon dioxide retention occurred because of insufficient alveolar ventilation during the procedure. This was largely a complication of the artificial respiration that is part of endotracheal positive pressure anesthesia. Since the present study was an attempt to define the physiologic effects of the surgical trauma itself and not of the anesthesia, a minimum of sixteen hours was allowed to pass after the completion of surgery before observations were made. This time interval was observed even in the patients undergoing thoracoplasty (in whom local anesthesia alone was employed) in order that the data would be comparable to the data collected upon patients in whom general anesthesia was to be employed.

No instance of carbon dioxide retention occurred in this series of cases studied after the three stages of thoracoplasty. The arterial blood pH and $P_{CO_2}$ were unchanged from the control values. The normal $P_{CO_2}$ in the postoperative period also indicates that the mild anoxemia which existed was insufficient to stimulate an increase in respiratory volume.

The degree of anoxemia which existed on the morning following surgery was slight, except in one case (G. J.) in whom atelectasis of the lower lobe occurred because of plugging of the bronchus by retained secretions. The fall from a mean of 95.79 per cent hemoglobin saturation to 90.66 per cent can largely be accounted for by the shunt of blood through recently collapsed lung tissue which was not being aerated. There was a 15.3 per cent shunt estimated to occur in the lung on the morning following surgery. The method used for this determination involved the breathing of 100 per cent oxygen by the subject and reflects only that blood passing through lung tissue which was not aerated at all. It does not demonstrate the quantity of blood passing through portions of lung which are ventilated to a less than normal degree. The effective shunt when the patient is breathing room air would, therefore, be even greater than these data demonstrate. The observation of a significant shunt of blood through recently collapsed pulmonary tissue is in agreement with the work of Berggren[10] in man and of Björk[11] in animals.

The observations herein reported throw light upon the respiratory adjustments made by the body after surgery to the chest. It has been shown in this series of cases that: (1) the total volume of ventilation per minute increased

The quantity of oxygen physically dissolved in the arterial blood by the exposure to an alveolar oxygen tension of about 600 mm. Hg fell from 1.78 volumes per cent preoperatively to 1.09 volumes per cent postoperatively. From this one can calculate that 0.69 volumes per cent fall occurred in the oxygen content of blood leaving the lungs. This change represents a 15.3 per cent absolute shunt through nonventilated lung tissue (Fig. 1*H*).

The minute volume of ventilation rose from a preoperative mean of 6,180 c.c. per minute to 7,234 c.c. per minute the morning following surgery (Fig. 2*A*). This change was accounted for primarily by an increased rate of respiration from 14.5 to 19.6 per minute (Fig. 2*B*). The tidal volume fell from a mean of 430 c.c. preoperatively to a mean of 390 c.c. postoperatively (Fig. 2*C*). The volume of the physiologic dead space changed only from a mean of 126.8 c.c. preoperatively to 135.6 c.c. in the immediate postoperative period (Fig. 2*D*). The volume of dead space that was ventilated each minute (physiologic dead space times respiratory rate) rose from a mean of 1,828 c.c. per minute preoperatively to 2,624 c.c. per minute postoperatively, i.e., a mean individual increase of 58 per cent above the preoperative level (Fig. 2*E*). Alveolar ventilation per breath decreased significantly from 257.2 c.c. before surgery to 207.9 c.c. shortly thereafter (Fig. 2*F*). The total volume of carbon dioxide excreted by the lungs per minute rose only from 217 c.c. per minute preoperatively to 230 c.c. per minute after surgery (Fig. 2*G*), in spite of the fact that the mean metabolic rate rose 19 per cent following surgery (Fig. 1*B*). This apparent discrepancy is accounted for by a significant fall in the mean respiratory quotient from 0.87 preoperatively to 0.78 postoperatively (Fig. 1*C*). The lower respiratory quotient following surgery was probably due to the poor nutrition on the day of surgery, a period of about twenty-four hours immediately preceding the observation. The volume of alveolar ventilation per minute (alveolar ventilation times respiratory rate) was increased proportionately from a mean of 3,685 c.c. per minute before operation to 4,008 c.c. per minute following surgery (Fig. 2*H*). The change in carbon dioxide excretion and alveolar ventilation per minute did not prove to be of statistical significance.

## DISCUSSION

The interest of this laboratory in the physiologic changes following thoracic surgery was aroused by the occurrence of unexplained shock in two patients early in 1949. The marked uncompensated respiratory acidosis which was found (arterial blood pH 7.10) was thought to be the basis for the clinical picture.

It is well known that many patients who require surgical therapy for pulmonary tuberculosis also have concomitant diffuse pulmonary disease such as emphysema and fibrosis.[2] The presence of an extensive diffuse pulmonary process of this type may lead to dyspnea during mild exertion or even at rest. Such a situation may preclude certain major operations on the chest. It was felt that an accurate determination of the stress placed upon the body in the postoperative period and of the efficiency with which the recently operated patient could meet this stress would be of value.

The foremost function of the lungs is the absorption of oxygen and the elimination of carbon dioxide. Of these, the carbon dioxide excretory factor is of the greater importance since oxygen is easily supplied therapeutically. The lungs are solely responsible for the elimination of carbon dioxide and thus play a vital role in the maintenance of the acid-base balance of the blood. There is as yet no way whereby the excretion of carbon dioxide can be facilitated save by an increased alveolar ventilation. Furthermore, for practical purposes, sole reliance for such ventilation must be placed upon the natural mechanisms of the body. If these mechanisms prove insufficient, carbon dioxide retention and respiratory acidosis ensue.

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The observations herein reported throw light upon the respiratory adjustments made by the body after surgery to the chest. It has been shown in this series of cases that: (1) the total volume of ventilation per minute increased appreciably; (2) the respiratory rate rose significantly; (3) the amount of carbon dioxide formed per minute rose slightly; (4) the respiratory quotient fell postoperatively.

From these facts certain deductions can be made relative to the physiologic mechanisms involved in the postoperative adjustment. The maintenance of alveolar ventilation was a necessary response to the continued formation of carbon dioxide at about the same or slightly higher level. If this had not occurred carbon dioxide retention and respiratory acidosis would have resulted. Pain limited the chest expansion possible per breath and thus decreased the tidal volume. A fall in tidal volume necessarily results in a decrease in alveolar ventilation achieved by each breath, since the volume of physiologic dead space cannot diminish. Actually, the physiologic dead space may be increased by rapid breathing because of the quicker movement of air through the respiratory bronchioles. In order to maintain adequate alveolar ventilation in the face of a smaller alveolar ventilation achieved by each breath, an increase in the rate of respiration occurred. Such an increase in rate with a smaller tidal volume constitutes an inefficient means of achieving adequate alveolar ventilation, since with every breath energy is wasted to move about 150 c.c. of air back and forth in the physiologic dead space. That this is of importance in the patients studied is shown by the great increase (58 per cent) in the volume of dead space ventilated per minute.

The data afford an explanation for Cournand's observation[2] that patients whose maximum breathing capacity was less than six times greater than their resting volume of ventilation (breathing reserve of less than 85 per cent) did not tolerate thoracoplasty satisfactorily. It has been shown that there is a definite and prolonged (several days) need for an increased minute volume of ventilation following thoracic surgery and that this must be accomplished by more rapid respiration. Patients who are unable to maintain this increased rate and volume of respiration would develop carbon dioxide retention which is a serious complication. A patient who is not able to achieve a sixfold increase over his volume of resting ventilation by a twenty- to thirty-second effort might well not be able to meet the prolonged need for a more rapid respiratory exchange after thoracic surgery.

It should be emphasized that the observations herein reported were made at a time when all direct effects of limited respiratory exchange during surgery would have been compensated and at a time when there was little increase in the rate of carbon dioxide excretion. The respiratory changes noted would, of course, be greatly exaggerated by the existence of acidosis with its attendant need for a greater alveolar ventilation. Such would be the situation in the respiratory acidosis at the close of intra-thoracic procedures, as observed by Beecher[16] and by Taylor.[17] The physiologic adjustments to such circumstances are presently being investigated and will be reported in a later paper.

From the studies reported in this paper it can be stated that if the $P_{CO_2}$ is found to be high on the day following thoracoplasty a diagnosis of pulmonary insufficiency is justified and steps should be taken to increase the volume of alveolar ventilation. This can probably best be done by the performance of a tracheotomy. By lowering the physiologic dead space this procedure allows a greater alveolar ventilation to be achieved by the same or smaller tidal volume of ventilation. The tracheotomy opening also presents less resistance to the exchange of air in and out of the lungs and, therefore, lessens the paradoxical motion of the flaccid chest wall.[18] Both of these effects help to produce a more efficient respiratory mechanism. The effect of tracheotomy in such cases is currently under investigation in this laboratory.

## SUMMARY AND CONCLUSIONS

1. The immediate physiologic postoperative effects of three stage, seven-rib thoracoplasty were studied by stages in ten men ranging in age from 23 to 62 years. Careful preoperative evaluation of pulmonary function was done in each case. Clinically the patients varied from men who were normal to men who were dyspneic on slight exertion.

2. A significant, but rather mild, degree of anoxemia was found to follow surgery in each instance. In one case the anoxemia was more severe but in this one there was coexisting atelectasis.

3. There was evidence of the passage of blood through the pulmonary vascular bed without exposure to alveolar air. This pulmonary shunt was probably due to continued perfusion of recently collapsed pulmonary tissue and accounted for most of the anoxemia found.

4. The postoperative increase in metabolic rate was accompanied by a proportionately greater increase in respiratory minute volume. An increase in the volume of dead space ventilated per minute accounted for most of the rise in the respiratory minute volume while the volume of alveolar ventilation increased only slightly. A fall in the respiratory quotient following decreased nutrition on the day of surgery explains the lack of a proportionate increase in the carbon dioxide eliminated in the presence of an elevated metabolic rate.

5. The nature of the stress thrown upon the organism by thoracoplasty may be summed up as follows: A chest whose expansion is limited by pain following surgical manipulation is required to maintain a slightly greater alveolar ventilation than it did preoperatively. This demand is met by an increased respiratory rate.

The authors are indebted to Miss Shirley Lundgren and Mr. John Ognanovich for valuable technical assistance.

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# CIRCULATORY AND RESPIRATORY EFFECTS OF CONTINUOUS VERSUS PHASIC LUNG INFLATION IN OPEN-CHEST DOGS

## Description of an Apparatus for the Application of Positive Pressure During Inspiration Only

Howard A. Frank, M.D., Alvin M. Cahan, M.D., and Henry Banks, M.D.
Boston, Mass.

THIS study was undertaken to evaluate the degree of circulatory and respiratory disturbance which may be produced by continuous lung inflation as conventionally employed during thoracic surgery, and the extent to which such adverse effects may be diminished by a return to atmospheric pressure in the trachea during expiration.

A device which applies positive pressure during inspiration only was designed and built by one of us (A. M. C.). Its purpose is to minimize the physiologic disturbance inherent in positive pressure respiration, rather than to improve surgical convenience by controlling or suspending spontaneous respiratory movement. The apparatus not only removes the impediment to expiration, but allows the subject to initiate inspiration, so that the imposition of an arbitrary pattern of respiration is avoided.

### METHODS

Healthy dogs premedicated with morphine sulphate (15 mg. intramuscularly), were anesthetized with ether until the lid reflex was lost. An effort was made to maintain this level of anesthesia throughout the experimental period. A tracheal cannula was placed, and a standard closed system established, permitting rebreathing through a freshly filled soda-lime cannister. The breathing bag was replaced by a cylindrical rubber bellows of several liters' capacity enclosed in an airtight glass cylinder. A vent in the cylinder maintained atmospheric pressure about the bellows, or else served to apply positive respiratory pressure. Tidal exchange was indicated by the excursion of the shadow of a wire mounted horizontally on the exterior of the glass cylinder projected to a vertical scale at the bottom of the bellows. Oxygen consumption was measured by noting the time required for the bellows to return to the null point after the introduction of 300 c.c. of oxygen by means of a water displacement apparatus. Dry gas volumes were corrected to standard conditions of temperature and pressure. Duplicate determination of oxygen consumption agreed within 3 per cent.

Mean respiratory pressures were indicated by a water manometer attached to a side-arm of the tracheal cannula. One femoral artery was exposed for blood samples, and the other was cannulated and attached to a mercury manometer. A catheter was introduced into the right auricle via the left internal jugular vein (position checked at end of experiment) for pressure measurements and mixed venous blood samples. Central (right auricular) and peripheral (femoral) venous pressures were measured by saline manometers, the zero point being set at the estimated level of the auricle.

Cardiac output was determined from oxygen consumption and arteriovenous blood oxygen difference by the Fick principle, and is expressed as "cardiac index" (L./min./m²).

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Surface area was calculated from the weight.[1] Blood oxygen and $CO_2$ content were determined in duplicate by the manometric method of Van Slyke and Neill.[2]* Arterial oxygen saturation was calculated after determining oxygen capacity by equilibration in a tonometer or in a few instances by calculation from hemoglobin concentration (Evelyn colorimeter). The hematocrit of each sample was measured. Arterial blood pH was determined by the Beckman potentiometer, using an enclosed glass electrode. The $CO_2$ pressure and buffer base concentration were obtained from the nomogram of Singer and Hastings.[5] Electrocardiograms were taken from standard limb leads using needle electrodes.

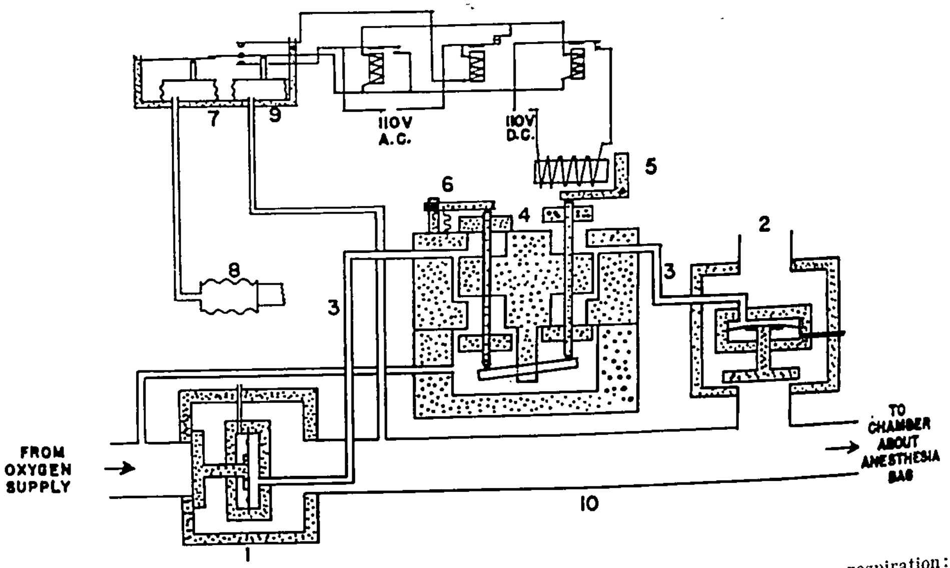

Fig. 1.—Schematic diagram of apparatus for inspiratory positive pressure respiration: the apparatus is designed to compress an enclosed anesthesia bag or bellows during inspiration and to allow a rapid return to atmospheric intratracheal pressure during each expiration. The compressing force is supplied by oxygen from a cylinder, the supply pressure having been reduced to a peak of 20 cm. water by a standard reduction valve and a type A-16 AAF spring-loaded demand regulator (not shown in the figure). Oxygen from this source provides the mechanical force required to open the inflow valve (*1*) and to close the exhaust valve (*2*) during inspiration. The gas pressure is applied to one or the other valve diaphragm (larger than the effective areas of the valve faces) by one of a pair of small ducts (*3*) leading from a set of reciprocal pilot valves (*4*). The pilot valves are brought to the inspiratory position by the action of an electromagnet (*5*), and are returned to the expiratory position by a spring (*6*). The magnet (direct current) is activated by a relay and holding circuit set in motion by the contraction of a metal bellows (*7*), which shortens as the rubber bellows (*8*) about the rib cage elongates at the start of inspiration. Expiration is initiated at the predetermined peak pressure by the elongation of a second metal bellows (*9*) in continuity with the main respiratory conduit (*10*). The electrical contact established by this second bellows (*9*) in its lengthened state breaks the circuit which has been supplying the electromagnet during inspiration, and allows the pilot valves to reverse to the expiratory position, so that the inflow valve (*1*) is closed by oxygen pressure, and the exhaust valve (*2*) is opened by the intracheal pressure, assisted by a spring (not shown) within the valve. The anesthesia bag then remains at atmospheric pressure until the next inspiration is begun.

The dogs were loosely held supine, and in each a chest wall incision was made which exposed the intercostal muscles along the full length of the right sixth interspace. Blood loss was held to a minimum. Initial observations were made one hour later. This preparation, i.e., normal respiration with closed chest, was studied as a control. In two other groups of dogs, the thoracotomy was completed and the wound edges were retracted to open the thorax widely. In Group CPP (continuous positive pressure) the vent in the glass cylinder enclosing the bellows was attached to a 2-liter rubber bag inflated to provide a mean intratracheal pressure of 10 to 13 cm. of water. This maintained a lung volume which just filled the chest in the expiratory position, with very little tidal exchange. In Group IPP (inspiratory positive pressure) the cylinder enclosing the bellows was attached to the device which applied positive pressure during inspiration (peak pressure 18 cm. $H_2O$; mean pressure 2.5 to 5.0 cm. $H_2O$), and allowed passive expiration against atmospheric pressure. The lung volume was observed to reach approximately *one-half* of full expansion during inspiration and to fall one-quarter expansion in expiration. Tidal volume was approximately 500 cubic centimeters.

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*The method of blood gas analysis was not modified to correct for the presence of ether in the blood because the magnitude of this error is small at this very light plane of ether anesthesia[3] and a satisfactory modification in technique[4] was not then available. It is considered that no error of interpretation was introduced since the blood ether concentration was kept nearly constant in each animal after a prolonged equilibration period, and was a factor common to all of the experimental groups.

All animals in the IPP and control groups tolerated the experimental procedure well, and a full set of observations was obtained at zero time (one hour after completion of preparations), at 1 to 1.5 hours, and again at 2 to 2.5 hours. Several CPP experiments had to be concluded within 1.5 hours because of marked fall in cardiac output and arterial pressure, whereas others could be carried for 2.5 hours.

*Apparatus for Inspiratory Positive Pressure Respiration (Fig. 1).*—The dog's chest was encircled by a rubber cuff connected to a hydron bellows so that inspiratory motion of the chest shortened the bellows and closed an electrical circuit which set the control valves into the inspiratory position and provided a free flow of oxygen at a maximal pressure of 20 cm. $H_2O$. Expiration was begun when the intratracheal pressure reached 18 cm. $H_2O$. At this pressure the expansion of a second hydron bellows interrupted the electrical circuit, and the return of the valves to the expiratory position permitted passive exhalation against atmospheric pressure (Fig. 2).

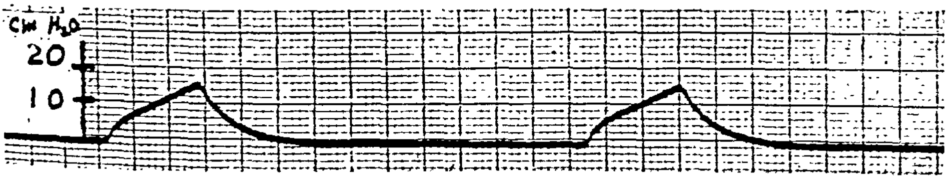

Fig. 2.—Sample intratracheal pressure curve during IPP respiration. An optical record taken from a membrane manometer attached to the intratracheal tube, converted to an electrical tracing by a pair of phototubes and inscribed by a direct-writing electrocardiograph. Calibrated by water manometer. Time interval between heavy vertical lines = 0.2 sec.

## RESULTS

A. *Circulatory Effects* (Tables I, *A*, *B*, *C*).—The initial determinations of cardiac index ranged from 3 to 12 L./min./m², the mean values for each group being close to 7.5 L./min./m² (Fig. 3).* By the end of the subsequent 2 to 2.5 hours of observation the cardiac outputs of the control dogs were lower and more closely grouped in the normal range (average 3.6 L./min./m²). Arterial pressures remained normal and the total procedure, including the blood sampling, was tolerated well.

In four of the nine CPP (continuous positive pressure) dogs, the cardiac index declined below 2.4 L./min./m² within 1.5 hours, and in three others after 2 to 2.5 hours. The cardiac index of two CPP dogs remained normal throughout the experimental period. The average cardiac index at the end of the experiments was 2.9 L./min./m² (43 per cent of the starting values). A reduction in arterial pressure, accentuated by the withdrawal of blood samples,

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*These high initial values may be attributed in part to the ether anesthesia.[6, 7]

TABLE I, A. CONTROL SERIES: DETERMINATIONS AT THE END OF THE PRELIMINARY EQUILIBRATION PERIOD (TIME ZERO) AND AT INTERVALS THEREAFTER

| DOG NUMBER | TIME (HOURS) | CARDIAC INDEX (L./MIN./$M^2$) | PH | $pCO_2$ (MM. HG) | BUFFER BASE (meq./L.) | ARTERIAL PRESSURE (mm. Hg) |
|---|---|---|---|---|---|---|
| 1. | 0 | 10.9 | 7.2 | 50 | 39 | 120 |
| | 1 | 13.9 | 7.20 | 44 | 37 | 120 |
| | 2 | 4.4 | 7.15 | 47 | 35 | 120 |
| 2. | 0 | 12.4 | 7.20 | 63 | 42 | 100 |
| | 1 | 6.4 | 7.20 | 59 | 41 | 100 |
| | 2 | 3.1 | 7.20 | 53 | 40 | 100 |
| 4. | 0 | 4.2 | 7.45 | 28 | 47 | 110 |
| | 1½ | 3.0 | 7.45 | 30 | 49 | 110 |
| | 2¼ | 3.8 | 7.40 | 33 | 46 | 110 |
| 5. | 0 | 4.4 | 7.18 | 38 | 36 | 90 |
| | 1 | 7.1 | 7.10 | 48 | 34 | 100 |
| | 2¼ | 2.7 | 7.10 | 41 | 32 | 100 |
| 6. | 0 | 5.7 | 7.20 | 40 | 36 | 120 |
| | 1¼ | 6.1 | 7.18 | 41 | 35 | 120 |
| | 2½ | 4.3 | 7.18 | 41 | 35 | 140 |
| Averages | 0 | 7.5 | 7.25 | 44 | 40 | 108 |
| | 1-1½ | 7.3 | 7.23 | 44 | 39 | 110 |
| | 2-2½ | 3.6 | 7.26 | 43 | 38 | 114 |

TABLE I, B. CPP SERIES: OPEN-CHEST DOGS BREATHING OXYGEN AT A PRESSURE SUFFICIENT TO MAINTAIN FULL LUNG VOLUME THROUGHOUT THE RESPIRATORY CYCLE

| | DOG NUMBER | TIME (HOURS) | CARDIAC INDEX (L./MIN./$M^2$) | PH | $PCO_2$ (MM. HG.) | BUFFER BASE (MEQ./L.) | ARTERIAL PRESSURE (MM. HG.) |
|---|---|---|---|---|---|---|---|
| Full lung inflation (Average respiratory pressure 10 to 13 cm. $H_2O$) | 1. | 0† | 7.8 | 7.25 | 45 | 42 | 100 |
| | | 1 | 2.3 | 6.85 | 130+ | 25 | 75 |
| | 2. | 0 | 6.8 | 7.25 | 56 | 46 | 100 |
| | | 1½ | 1.9 | 6.75 | 200+ | 10 | 110 |
| | 3. | 0 | 9.0 | 7.30 | 52 | 48 | 110 |
| | | 2 | 4.9 | 6.96 | 120 | 37 | 110 |
| | 4. | 0 | 4.3 | 7.50 | 38 | 54 | 90 |
| | | 1 | 3.4 | 7.40 | 45 | 50 | 90 |
| | 5. | 0 | 6.2 | 7.30 | 30 | 38 | 110 |
| | | 2½ | 5.2 | 7.05 | 45 | 28 | 110 |
| | 6 | 0 | 6.0 | 7.35 | 37 | 45 | 110 |
| | | 2 | 2.3 | 6.85 | 130+ | 25 | 110 |
| | 7. | 0 | 12.0 | 7.40 | 38 | 48 | 110 |
| | | 1½ | 1.6 | 7.10 | 63 | 36 | 70 |
| | 8. | 0 | 6.8 | 7.15 | 58 | 37 | 100 |
| | | ¼ | 5.0 | 7.02 | 91 | 36 | 100 |
| | | 1½ | 3.9 | 6.90 | 118 | 31 | 100 |
| | | 2½ | 2.2 | 6.80 | 140+ | 27 | 100 |
| | 11. | 0 | 6.5 | 7.45 | 34 | 48 | 100 |
| | | ¼ | 2.7 | 7.10 | 82 | 40 | 90 |
| | | 1 | 2.2 | 7.00 | 88 | 34 | 80 |
| | | 2 | 2.1 | 7.00 | 88 | 34 | 80 |
| Averages | (9 dogs) | 0 | 7.3 | 7.36 | 43 | 45 | 103 |
| | (6 dogs) | 1-1½ | 2.5 | 7.00 | 107+ | 31 | 87 |
| | (5 dogs)* | 2-2½ | 3.3 | 6.93 | 103+ | 30 | 102 |
| Lung half inflated (Respiratory pressure 4 cm. $H_2O$) | 9. | 0 | 8.5 | 7.25 | 53 | 43 | 100 |
| | | 1½ | 2.3 | 6.77 | 150+ | 23 | 180 |
| | 10. | 0 | 9.2 | 7.35 | 36 | 44 | 100 |
| | | 1½ | (dying) | 6.81 | 150+ | 24 | 180 |

*Four dogs did not tolerate CPP respiration beyond 1.5 hours.

†Time zero refers to the end of the preliminary equilibration period and the moment before open thoracotomy and positive pressure respiration were established.

accompanied the decline in cardiac output in some of the animals, but marked elevation in arterial pressure occurred when the $CO_2$ accumulation was very great (Experiments 9 and 10, Table I, B).

The IPP (inspiratory positive pressure) group (seven dogs) showed the least decline in cardiac output, to an average of 5.4 L./min./$m^2$, or 85 per cent of the initial indices. In only two did the cardiac index fall below 3 L./min./$m^2$, and in these two the arterial pressure also declined.

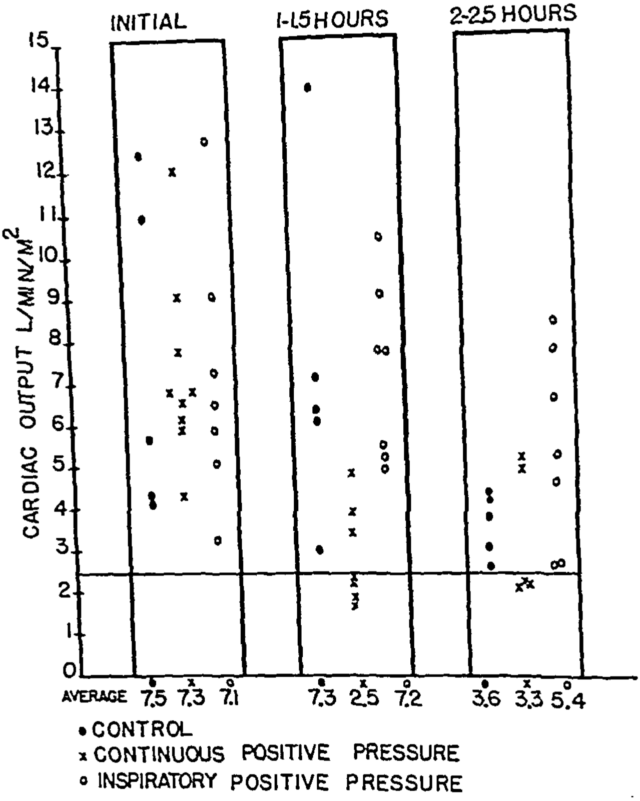

Fig. 3.—Cardiac output (L./min./$m^2$) in the three groups of dogs at three phases of the experiment. The control dogs retained intact pleural sacs and breathed at atmospheric pressure. Open thoracotomy and positive pressure respiration was established in the other two groups immediately after the initial determinations of cardiac output.

The horizontal line at 2.4 L/min/$m^2$ represents the lower limit of cardiac output in normal dogs.[8, 9] Four dogs in the CPP group collapsed within 1.5 hours and are not represented in the third column. The two CPP dogs showing normal cardiac outputs at 2 to 2.5 hours are not represented in the 1 to 1.5 hours column. (CPP dogs 9 and 10, whose lungs were only half inflated, are not included in Figs. 3, 4, or 5.) All dogs in the control and IPP groups are represented in all three columns.

The initial femoral-auricular venous pressure gradient ranged from 1.0 to 6.5 cm. $H_2O$ (average 4.0 cm. $H_2O$). Auricular and femoral venous pressure did not change during control observation. An elevation in both central and peripheral venous pressure occurred as soon as the chest was opened and positive respiratory pressure was applied (Table II). The new levels of venous pressure were reached within three to five minutes and did not change thereafter unless

TABLE I, C. IPP SERIES: OPEN-CHEST DOGS BREATHING OXYGEN INTERMITTENTLY SUPPLIED SO AS TO INFLATE THE LUNGS DURING INSPIRATION ONLY

| DOG NUMBER | TIME (HOURS) | CARDIAC INDEX (L./MIN./M²) | PH | $PCO_2$ (MM. HG.) | BUFFER BASE (MEQ./L.) | ARTERIAL PRESSURE (MM. HG.) |
|---|---|---|---|---|---|---|
| 4. | 0† | 7.3 | 7.35 | 39 | 44 | 100 |
|  | 1 | 5.5 | 7.40 | 27 | 42 | 80-100 |
|  | 2¼ | 2.7 | 7.40 | 22 | 38 | 80 |
| 5. | 0 | 5.9 | 7.48 | 34 | 52 | 100 |
|  | 1 | 7.7 | 7.35 | 48 | 49 | 100 |
|  | 2 | 5.2 | 7.23 | 63 | 45 | 100 |
| 7. | 0 | 3.3 | 7.50 | 32 | 52 | 110 |
|  | 1 | 4.9 | 7.50 | 40 | 56 | 110 |
|  | 2¼ | 6.6 | 7.50 | 40 | 56 | 110 |
| 8. | 0 | 5.1 | — | — | — | 110 |
|  | 1 | 9.1 | 7.50 | 27 | 49 | 100 |
|  | 2 | 2.7 | 7.40 | 28 | 40 | 60 |
| 9. | 0 | 6.5 | 7.20 | 56 | 42 | 100 |
|  | 1 | 5.2 | 7.20 | 50 | 40 | 100 |
|  | 2 | 4.5 | 7.20 | 52 | 41 | 100 |
| 10. | 0 | 9.1 | 7.35 | 39 | 46 | 100 |
|  | 1 | 7.7 | 7.40 | 39 | 50 | 100 |
|  | 2 | 7.7 | 7.15 | 66 | 40 | 100 |
| 11. | 0 | 12.7 | 7.30 | 36 | 41 | 100 |
|  | 1 | 10.5 | 7.20 | 51 | 40 | 100 |
|  | 2 | 8.4 | 7.20 | 62 | 43 | 100 |
| Averages | 0 | 7.1 | 7.36 | 39 | 46 | 103 |
|  | 1 | 7.2 | 7.36 | 40 | 47 | 100 |
|  | 2-2¼ | 5.4 | 7.29 | 47 | 44 | 93 |

†Time zero refers to the end of the preliminary equilibration period and the moment before open thoracotomy and positive pressure respiration were established.

the respiratory pressures were altered. In the CPP group, the mean increase in auricular pressure was 5 cm. $H_2O$, and in the femoral pressure 4 cm. $H_2O$. In experiments 9 and 10 in which the lungs were only partly inflated the largest

TABLE II. RELATIONSHIP OF VENOUS PRESSURE CHANGE TO INCREASE IN RESPIRATORY PRESSURE DURING CPP AND IPP RESPIRATION IN OPEN-CHEST DOGS

| SERIES | DOG NUMBER | INCREASE IN RESPIRATORY PRESSURE (CM. $H_2O$) | INCREASE IN AURICULAR PRESSURE (CM. $H_2O$) | CHANGE IN FEMORAL-AURICULAR VENOUS PRESSURE GRADIENT (CM. $H_2O$) |
|---|---|---|---|---|
| *CPP* | 1. | 10 | 5.0 | –2.0 |
|  | 2. | 13 | 7.0 | 0 |
|  | 3. | 10 | 5.5 | 0 |
|  | 4. | 10 | – | – |
|  | 5. | 10 | 6.0 | –2.0 |
|  | 6. | 10 | 6.5 | –1.5 |
|  | 7. | 10 | 5.0 | –1.5 |
|  | 8. | 10 | 4.5 | –0.5 |
|  | 11. | 11 | 4.0 | –0.5 |
|  | Mean | 10 | 5.0 | –1.0 |
| (half-inflation) | 9. | 4 | 9.0 | +0.5 |
|  | 10. | 6 | 10.0 | –3.5 |
| *IPP* | 4. | 2.5 to 5.0 | 4.0 | 0 |
|  | 5. |  | 4.0 | –1.5 |
|  | 7. |  | 4.5 | –3.0 |
|  | 8. |  | 4.0 | –1.0 |
|  | 9. |  | 1.5 | +1.0 |
|  | 10. |  | 1.0 | +1.0 |
|  | 11. |  | 2.0 | +1.0 |
|  | Mean | 2.5 to 5.0 | 3.0 | 0 |

increase in central venous pressure, 9 to 10 cm. water, was reached. These are not included in this average. In the IPP group, the mean increase of 3 cm. $H_2O$ in central venous pressure was accompanied by an equal increase in femoral venous pressure. Changes in venous pressure gradient in individual dogs could not be correlated with changes in cardiac output.[*]

Study of the electrocardiograms in three leads disclosed no significant alteration in conduction, electrical activity or in the mechanism of the heart-beat.

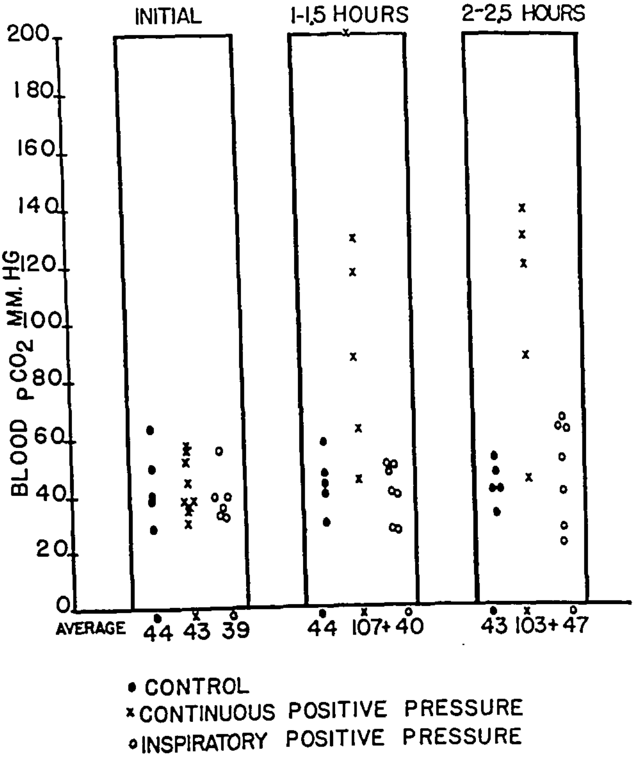

Fig. 4.—Arterial blood $pCO_2$ (mm. Hg) in the three groups of dogs at three phases of the experiment (as described in the legend of Fig. 3).

B. *Respiratory Effects* (Tables I, A, B, C).—Arterial oxygen saturation remained above 95 per cent in all dogs except for four in the CPP group in which the values lay between 87 per cent and 91 per cent.

Arterial $pCO_2$ (mean initial values 39 to 44 mm. Hg) remained unchanged in the control dogs, but rose to extreme values during CPP respiration (Fig. 4). The average arterial $pCO_2$ at the end of the period of CPP respiration was 107 mm. Hg in the nine dogs maintained at full lung inflation, and was 150 mm. Hg in the two dogs whose lungs were half inflated. During IPP respiration, in contrast, there was little or no $CO_2$ accumulation.*

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[*] The pressure determined in any selected vein is but a sample of a "peripheral venous pressure," and cardiac output cannot be expected to be strictly proportional to changes in femoral vein—right auricular gradient. Furthermore, the pressures recorded are mean pressures, so that a zero gradient need not mean zero flow. Forward flow will continue in those phases in which the gradient is positive, while backflow during phases of negative gradient is prevented by the venous valves.

Acidosis of severe degree developed regularly during CPP respiration (Fig. 5). The arterial pH dropped from the mean initial value of 7.36 to a mean of 6.97 by the end of the period of CPP respiration. In one dog the pH dropped from 7.45 to 7.10 and the $pCO_2$ rose from 37 to 82 mm. Hg within fifteen minutes of CPP respiration. Another animal reached a pH of 6.75 and a $pCO_2$ above 200 mm. Hg within one and one-half hours. In most animals metabolic acidosis accompanied the respiratory acidosis which developed during CPP respiration (Fig. 6).† The average buffer base level declined from the control value of 45 meq. per liter to 30 meq. per liter by the end of the experimental period. In no instance was there evidence of an increase in concentration of fixed base which might have been interpreted as a compensatory change in response to respiratory acidosis. The degree of decline in cardiac output could not be correlated with the degree of acid-base disturbance.

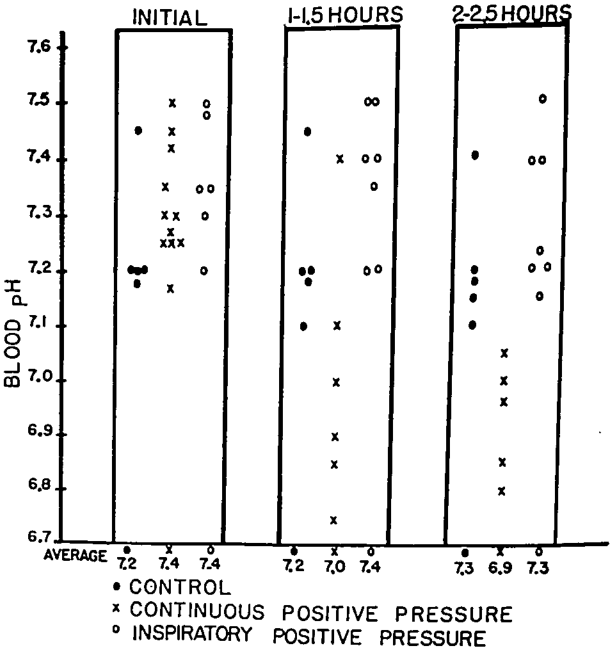

Fig. 5.—Arterial blood pH in the three groups of dogs at three phases of the experiment (as described in the legend of Fig. 3).

IPP respiration throughout the period of open-chest observation prevented any significant change in blood pH.

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*The IPP instrument can produce marked overventilation and $CO_2$ depletion if not controlled by the dog's respiratory rhythm. In two discarded experiments an electrode attached to a nostril was used to initiate inspiration. This proved to be oversensitive to extraneous movement and overbreathing was produced, so that arterial $pCO_2$ fell below 11 mm. Hg. and the arterial pH rose to 7.6. The cardiac outputs of these two dogs remained normal.

†Mild metabolic acidosis was observed in a number of dogs at the time of the initial blood sample. Metabolic acidosis may be attributed to ether anesthesia in dogs,[11, 12] although apparently not in man.[13]

In three supplementary experiments an effort was made to correct the respiratory acidosis of CPP respiration by brief periods of repeated emptying and filling of the lungs, such as might be done manually by an anesthetist. In D1, the lungs were allowed to empty and were then reinflated; this cycle was repeated four times in immediate succession. Emptying proceeded so slowly that ventilation was further impaired rather than improved, and the acidosis which had been mild (pH 7.2, $pCO_2$ 45 mm. Hg) was intensified (pH 7.05, $pCO_2$ 70 mm. Hg). In D2, after an hour of CPP respiration, the pH was 7.0 and the $pCO_2$ 106 mm. Hg. During a subsequent ten-minute period of IPP respiration, using the device herein described, these values remained unchanged. In D3, CPP respiration was interrupted and the lungs partially deflated and inflated twice every fifteen minutes. Nevertheless, severe respiratory acidosis developed within an hour (pH 6.92, $pCO_2$ 116 mm. Hg).

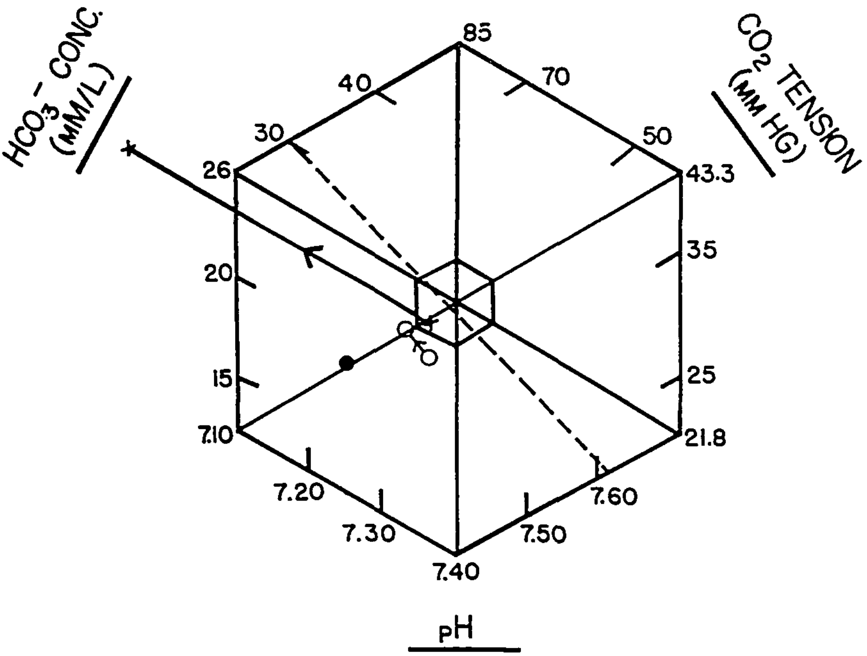

Fig. 6.—Triaxial diagram of pH, $pCO_2$, and bicarbonate concentration, adopted from Hastings and Steinhaus.[10] The broken line indicates the path of uncomplicated $CO_2$ increase. The solid circle represents the average of control values. No change occurred during control observation. The open circles indicate the average of values before and after IPP respiration. The change is small. The crosses indicated the values before and after CPP respiration. Marked acidosis and $CO_2$ accumulation are denoted. The deviation of the CPP path to the left, with reference to the broken line of uncomplicated $CO_2$ accumulation, indicates the metabolic component.

## DISCUSSION

In intact animals undergoing positive pressure respiration, reduced ventilation is less critical than the interference with circulation consequent to elevation of intrapulmonary and intrapleural pressure.[14-30] In the open-chest animal, however, the rise in intrapleural pressure is limited, so that the decline in ventilation is the most obvious adverse effect of continuously maintained positive respiratory pressure. But cardiac output also falls and to a greater degree than might be expected on the basis of the intrapleural pressure change alone.[31-33] Since the decrease in cardiac output is greater during CPP (continuous positive pressure) respiration than during IPP (inspiratory positive pressure) respiration, the level of intrapleural pressure, which is presumably the same in both (open-chest), is not the only controlling factor. Hence the mean level of intrapulmonary pressure also influences cardiac output in these circumstances. An increase in intrapulmonary vascular resistance[16, 34] is suggested by the greater increase in right auricular pressure during CPP respiration.*

The CPP animals might have been expected to have the higher cardiac outputs because of the exaggerated respiratory effort accompanying $CO_2$ accumulation, and because acidosis as well as $CO_2$ accumulation are said to increase cardiac output.[36, 37] The observed contrary effect is not attributable to a greater degree of tachycardia[38] nor was there evidence of plasma volume loss[39] or inadequacy of "effective venous pressure."† Since respiratory motion produces blood flow even in the absence of heartbeat,[42] its absence during CPP respiration may partially account for poorer circulatory function than during IPP respiration. Except for this pumping action, the favorable effect of IPP respiration upon the circulation should probably be attributed to the lower mean intrapulmonary pressure, rather than to the intermittency of the positive pressure application.

The arterial pressures observed during CPP experiments were presumably the resultant of the opposing effects of high $CO_2$ tension[43] and reduced cardiac output. Poor tolerance of blood sampling was manifested by animals with low cardiac output, whether the arterial pressure was normal or low.

Open-chest animals with lungs maintained in continuous inflation (CPP) have so little tidal exchange that the elimination of $CO_2$ is inadequate. Severe respiratory acidosis results, but the cause of the accompanying metabolic acidosis is obscure. The lowered cardiac output may produce tissue anoxia and reduced renal function, with a resulting accumulation of lactate, pyruvate, phosphate, and sulfate ions, but the reduction in buffer base is greater than the observed degree of peripheral vascular disturbance can account for.[44]

Allowing the lung to collapse completely and then refilling it with oxygen is not an effective technique of clearing respiratory acidosis and may make it worse. Periodic partial emptying and reinflation of the lung at intervals of fifteen to thirty minutes, such as is commonly done by the anesthetist during open-chest surgery, does not prevent $CO_2$ accumulation. Even a return to effective ventilation does not correct respiratory acidosis as rapidly as it develops. This may be because intracellular changes which have occurred during the period of $CO_2$ accumulation are not immediately responsive to reduction in alveolar $CO_2$ tension. A similar delay in improvement, indeed an initial increase in acidosis, has been observed during resuscitation following experimental asphyxia.[45] Phasic interruption of the applied respiratory pressure to permit free exhalation (IPP) maintains effective ventilation throughout the open-chest period.

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*However, partial collapse of the lungs also produces high pulmonary vascular resistance.[35] The highest auricular pressure in the current experiments were found in the two dogs (CPP 9 and 10) whose lungs were allowed to remain half collapsed at low respiratory pressure.

†The "effective venous pressure" is defined[40] as the pressure available for filling the relaxed ventricle and is the gradient from auricular to extracardiac (intrapleural) pressure[41] This was above the critical limit for right ventricular output (5 cm. $H_2O$)[40] in all experiments.

The human subject appears to withstand prolonged open-chest surgery under a form of positive pressure respiration which is poorly tolerated by the dog. With a unilateral thoracotomy he ventilates somewhat better than the dog, in which the flimsiness of the anterior mediastinum prevents any expansion of the contralateral lung. This advantage may[46] or may not[47] be reduced in the lateral position, in which expansion of the intact hemithorax is limited by the patient's weight. In the prone position a congested or relatively inelastic human lung requires little or no positive respiratory pressure to maintain partial expansion. This may reduce the disturbance of circulatory dynamics but it should not improve ventilation. It has recently been shown that a severe degree of respiratory acidosis may indeed develop within two hours of human open-chest surgery[48, 49] and that this is accompanied by an unexplained metabolic acidosis. Respiratory acidosis is less likely to occur when the patient is supine, or suspended by the shoulders and pelvis in the prone position so that the thorax bears no weight, than in lateral or simple prone position.[50, 51] The level of anesthesia also plays a part, for respiratory acidosis may be observed during deep narcosis for operations other than thoracotomy.[52] The transfusion therapy routinely applied to compensate for blood loss and surgical trauma probably also tends to correct the decline in circulation resulting from positive pressure respiration, but the degree of circulatory inadequacy which remains is not known. Elevation in arterial pressure produced by the increase in $CO_2$ tension may give a falsely favorable impression of the state of the circulation. The ordinarily transitory rise in pulmonary arterial pressure and fall in cardiac output accompanying ligation of a major pulmonary artery[53, 54] may be accentuated by positive pressure respiration. The human heart is less able to tolerate severe acidosis than is the dog's heart, as judged by electrocardiographic evidence.[55] Our studies agree with those of others in not finding ECG changes in the CPP dogs,[55, 56] but evidence of myocardial damage has been found to accompany severe $CO_2$ acidosis in well-oxygenated patients.[55] Respiratory acidosis during cardiac surgery has been reported to increase susceptibility to acute arrhythmias.[57] $CO_2$ accumulation produces a marked rise in cerebrospinal fluid pressure.[58] It is apparent, then, that continuous lung inflation is not entirely satisfactory for clinical intrathoracic surgery and may account for a number of adverse reactions.

Mechanically imposed ventilation is applied during open-chest surgery in a number of clinics.[59-63] Manual compression and release of the breathing bag throughout the procedure may prevent respiratory acidosis.[50] Controlled respiration, whether accomplished manually or by automatic device, in the absence of continuous and precise monitoring of arterial blood $CO_2$ pressure, pH and oxygen tension is unlikely to be as responsive to the patient's needs as spontaneous respiration. The apparatus herein described is under the control of the subject's respiratory center. The data presented indicate, for dogs at least, that the performance of the respiratory center remains satisfactory under light ether anesthesia.

## SUMMARY

Continuous inflation of the lungs of open-chest dogs for a period of two hours or less is usually accompanied by a substantial decline in cardiac output and the development of respiratory acidosis of severe degree. These adverse effects may be minimized or avoided by interrupting the positive respiratory pressure to permit passive exhalation against atmospheric pressure. A device is described which applies positive respiratory pressure during inspiration only, and which is controlled by the animal's own respiratory rhythm.

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# SUCCESSFUL REMOVAL OF TUMOR OF THE HEART

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TUMORS of the heart, both primary and metastatic, are comparatively rare. According to an excellent review of the subject by Prichard[1] in January, 1951, approximately 500 metastatic tumors of the heart and 415 primary ones have been reported in the literature. Of the primary tumors, 112 were sarcomas, and 303 were benign lesions. According to Prichard,[1] who quotes from Martin's report of 1929, only twenty-one lipomas are included in the series of benign primary cardiac tumor cases. A review of Martin's[2] publication, however, reveals that only one incidence of lipoma of the heart was presented therein by the author. Twenty additional cases of lipoma were referred to in the literature, but were not confirmed by bibliographical references.

A second case report of lipoma of the heart, in a 7-month-old baby girl, was described by Brewis[3] in 1905. At necropsy, a "fatty tumor" was encountered arising from the interventricular septum. The tricuspid valve was obstructed by the portion of tumor which protruded into the right ventricle. As accurately as could be determined, only two proved cases of lipoma of the heart have been reported. No lipoma of the heart has been diagnosed ante mortem, nor has one been successfully removed by operation. Of the 303 benign tumors of the heart reported in the literature, only five were diagnosed before death of the patient.

Successful surgical removal of a cardiac tumor is exceedingly rare. Only one instance has been recorded by Beck[4] in 1942. Two patients, according to Beck, were operated upon successfully, but in only one instance was a true tumor removed. The first operation was performed in two stages, in 1934 and 1936, upon a white man 22 years of age. An intrapericardial teratoma, which arose from the right ventricle and was intimately adherent to the aorta, pulmonary artery, vena cava, and right inferior pulmonary vein, was successfully removed. The second operation was performed by Beck in 1940 upon a 39-year-old white man. A calcified cyst, without any pathologic evidence of true neoplastic formation, was removed from the left ventricular wall. Both patients survived and did well after surgery. No other reports of the successful removal of a cardiac tumor have been encountered in this author's review of the literature.

In 1941-1942, Gebauer[5] attempted, in three stages, operative removal of a teratoma and cyst from the heart of a 12-year-old girl. Unfortunately, the patient expired on the operating table because of uncontrollable hemorrhage from a perforation in the aorta, produced inadvertently during the application of a clamp across the tumor pedicle.

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The following case report is presented because of the extreme rarity of the lesion involved, the unusually large size of the tumor (3½ pounds), and because it is the first recorded instance of successful surgical removal of a primary lipoma of the heart.

## CASE REPORT*

*C. D.*, A 44-year-old white man, was first seen by Dr. Henry F. Kulle of Cincinnati, because of bleeding from the rectum, easy fatigability, and vague discomfort over the anterior chest. There was no history of cardiac decompensation, cough, hemoptysis, or remote pulmonary disease. Fluoroscopic examination of the patient's chest by Dr. Kulle showed the cardiac silhouette to be greatly enlarged in all directions.

The patient was admitted for further study to the Good Samaritan Hospital of Cincinnati on July 10, 1951.

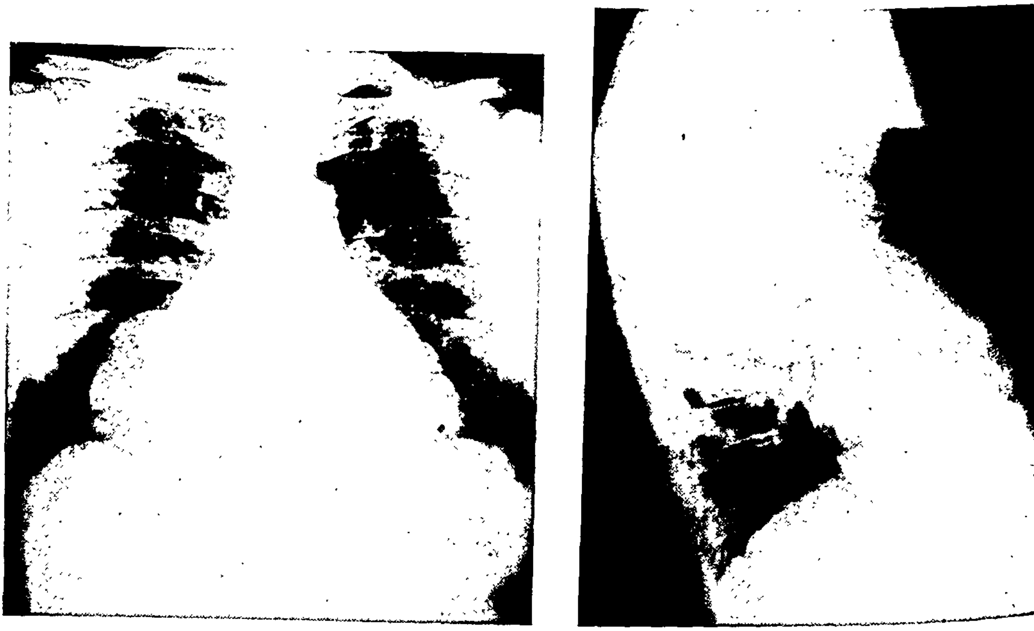

Fig. 1. Fig. 2.

Figs. 1 and 2.—Posteroanterior and lateral projection roentgenograms showing enlargement of the cardiac silhouette.

*Physical Examination.*—The patient was a well-developed, moderately obese white man, who did not appear to be in any acute or chronic distress. The temperature was normal, blood pressure was 110/80, and the pulse 85 per minute. No arrhythmias nor paradoxical pulses were noted. There was no distention of the neck or thoracic veins, which would suggest any elevation in the venous pressure. The arm veins emptied rapidly on elevation. The lungs were entirely clear to percussion and auscultation, and the breath sounds were somewhat distant. No adventitious sounds were audible. The heart sounds were of good quality, but were somewhat muffled and distant. The apex impulse was difficult to locate by palpation. There seemed to be very marked enlargement of the cardiac diameter by percussion. The rest of the physical examination revealed entirely normal findings.

*Roentgenograms.*—Preliminary roentgenograms of the chest, posteroanterior and right lateral projections (Figs. 1 and 2), showed enlargement of the cardiac silhouette in all dimensions, particularly to the right of the midline. Barium studies of the entire gastroenteric tract revealed no evidence of organic lesions.

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*The author is indebted to Dr. Henry F. Kulle for permission to publish this case report.

An electrocardiogram made on July 12, 1951, revealed essentially normal findings, except for prolongation of the auriculoventricular conduction time to a high normal, and moderate left axis deviation. The tracing was thought to be within normal limits.

Laboratory examinations, including urinalysis, complete blood cell counts, and hemoglobin determinations, revealed normal findings.

An angiocardiogram (Fig. 3) performed on July 11, 1951, with 70 per cent diodrast, showed excellent filling of the inflow tract, including the superior vena cava, right auricle, and a portion of the right ventricle. Some dye was also seen in the left side of the heart. The greater portion of the density seen on the posteroanterior projection roentgenogram of the chest appeared to be due to an extracardiac tumor or cyst, which was not attached to the heart. A provisional diagnosis of pericardial coelomic cyst was made. Twenty-four hours preceding operation, the patient received S. R. penicillin, 400,000 units, and one-half gram of streptomycin hypodermically, every twelve hours.

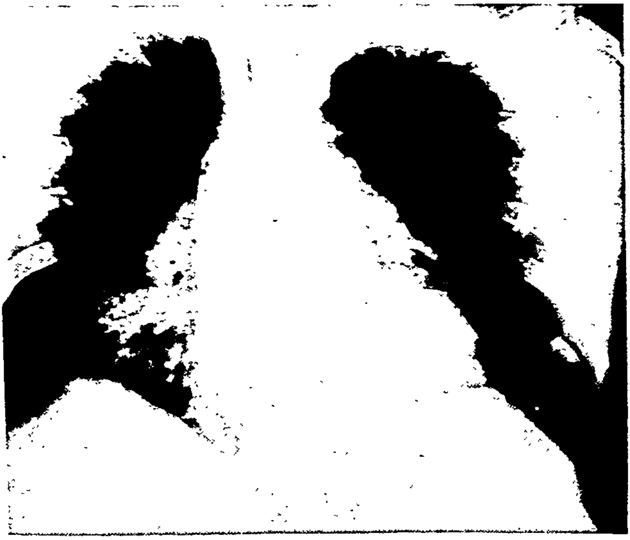

Fig. 3.—Angiocardiogram showing excellent filling of the inflow tract and the extracardiac location of the tumor.

*Operation.*—July 14, 1951, at Good Samaritan Hospital. With the patient in the left lateral prone position and under endotracheal gas, oxygen, and ether anesthesia, a curved periscapular incision was made over the right side of the thorax. The sixth rib was subperiosteally resected in its entirety, and the pleural cavity was entered through the rib bed. The lower and middle lobes of the right lung were adherent to the pericardium and diaphragm by numerous broad adhesions, which were divided by sharp dissection. The pericardium was extremely thin, transparent, and distended to approximately ten times normal dimensions, so that it bulged into the right chest. A normal-sized phrenic nerve coursed in the usual anatomic position, over the outside of the pericardial sac. Through the thin, transparent pericardial wall, numerous coils of yellowish gray tissue could be seen, which on first inspection looked not too much unlike loops of intestine. As soon as it was determined that manipulation of the heart would be necessary, the patient was given 20 c.c. of 1 per cent procaine by intravenous drip, to decrease the sensitivity of the heart, and to prevent arrhythmias during manipulation. A longitudinal incision was made in the pericardial sac anterior to the phrenic nerve, and the sac was opened widely. Almost immediately, numerous large loops and coils of adipose tissue bulged into the right chest. The entire tumor was completely delivered down to a common stalk, which was attached to the anterior surface of the left ventricle, over a circular area which measured approximately 4 by 4 cm. in diameter. At this point in the operation, it was obvious that the lesion was a massive lipoma, arranged in loops and coils, and arising by a common pedicle from the left ventricular wall. The point of attachment of the lipoma to the ventricular wall was separated without too much difficulty, by alternate sharp and blunt dissection. As nearly as could be determined, the tumor arose from the superficial layer of the myocardium beneath the epicardium, and penetrated the myocardium for a distance of several millimeters. Following the separation of the tumor pedicle, there was a moderate amount of brisk bleeding from the surface of the heart. Persistent bleeding from one moderate-sized vessel was controlled with warm moist compresses, rather than suture ligature, because of the proximity of the bleeder to the coronary artery. Approximately 150 c.c. of blood accumulated, as a result of the bleeding from the amputation site. During the course of the operation, the chamber of the heart was not entered. Examination of the interior of the tremendously dilated pericardial sac showed it to be in continuity and without any connection with the abdomen or contralateral hemithorax. Grossly, the heart appeared to be entirely normal. The superior vena cava was not dilated and showed no evidence of previous obstruction.

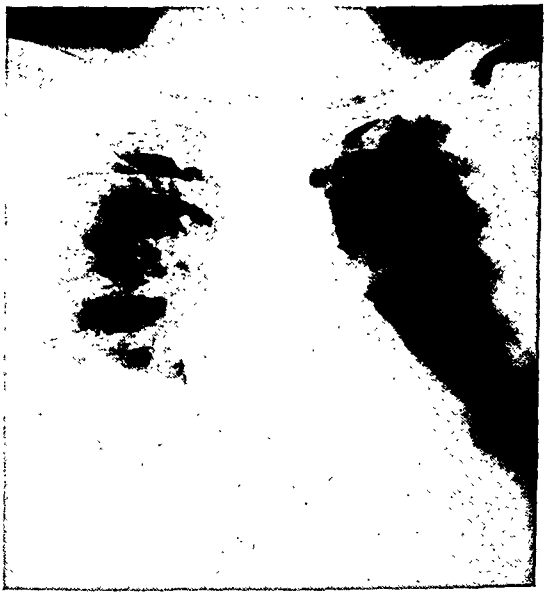

Fig. 4.—Posteroanterior projection roentgenogram of the chest showing return of the cardiac silhouette to normal size following removal of the tumor.

It was the author's impression that the easy distensibility of the pericardial sac and the slow growth of the tumor presumably accounted for the absence of venous obstruction and elevated venous pressure. The interior of the pericardial cavity was irrigated thoroughly with saline solution, and was then aspirated dry, following which procedure the incision in the pericardium was closed with interrupted sutures of medium silk. A small window, three to four centimeters in diameter, was allowed to remain in the inferior portion of the pericardial incision, to permit drainage of the pericardial cavity into the right chest. At this point in the operation it was noted that the right phrenic nerve had been inadvertently crushed by a clamp, which had been placed along the margin of the pericardial incision for retraction. This resulted in partial paralysis of the right, diaphragmatic leaf. The right chest was lavaged with saline solution, and was then aspirated dry, following which procedure the right lung was re-expanded by positive pressure and manipulation. The chest wound was closed by interrupted medium silk sutures, which were applied to the periosteal rib bed, intercostal muscles, chest wall, muscles, fascia, and skin. A dry sterile dressing was then applied, and the patient was returned to his room in good condition.

Following the operation the patient had a very uneventful course. He was ambulatory by his second postoperative day, and was discharged from the hospital, with the wound healed, on the ninth postoperative day.

When seen in a follow-up examination, two and one-half months after the operation, the patient was entirely without complaints, except for some discomfort over the anterior portion of the chest wound. There was no complaint of dyspnea, palpitation, or precordial discomfort. A roentgenogram of the chest made two months following the operation (Fig. 4) showed the cardiac silhouette to be approximately normal in size. The right lung was completely re-expanded, and there was no evidence of effusion. The right diaphragm remained partially elevated, as a result of the phrenic nerve crush at the time of operation.

*Pathology.*—Grossly, the tumor consisted of numerous coils and loops of fat tissue, which arose from a common stalk or pedicle (Fig. 5). The fresh specimen weighed 3.5 pounds. Microscopic examination revealed typical fat cells, with irregular limits (Fig. 6). There was no evidence of malignant degeneration in any portion of the tumor.

*Diagnosis.*—Lipoma.

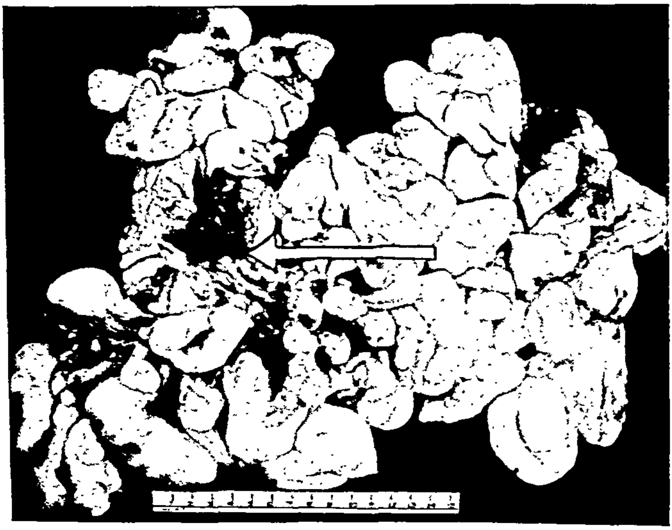

Fig. 5.—Photograph of gross specimen. Arrow indicates point of attachment of tumor to the heart.

## DISCUSSION

Primary lipoma of the heart is a pathologic rarity. In a review of the literature only two authenticated cases are recorded. There seems to be no record in the past of successful surgical removal of a cardiac lipoma. A study of the case reports indicates that this is the largest primary tumor of the heart ever reported, including all varieties.

A more accurate preoperative diagnosis could possibly have been made by utilizing a diagnostic pneumopericardium, as suggested by Leubschitz, Lundsteen, and Forchhammer.[6] The use of a preoperative diagnostic pneumopericardium was discussed but finally decided against, because of the history and angiocardiographic findings, which suggested a pericardial coelomic cyst. In retrospect, an artificially induced pneumopericardium before the operation would undoubtedly have settled the issue.

Not encountered in the case reported were certain classical symptoms and signs of tumor of the heart, such as decompensation developing without demonstrable cause and proving absolutely refractory to treatment, arrhythmias, failure of conduction as demonstrated on the electrocardiogram, and hemorrhagic pericardial effusion.

The prognosis in cardiac tumors is bad, as a rule, even in the case of benign tumors. There are, however, cases on record, in which symptoms of intracardiac myxomata were present for many years, as in the example reported by Strouse[7] in 1938. The duration of the disease in this instance was forty-three years.

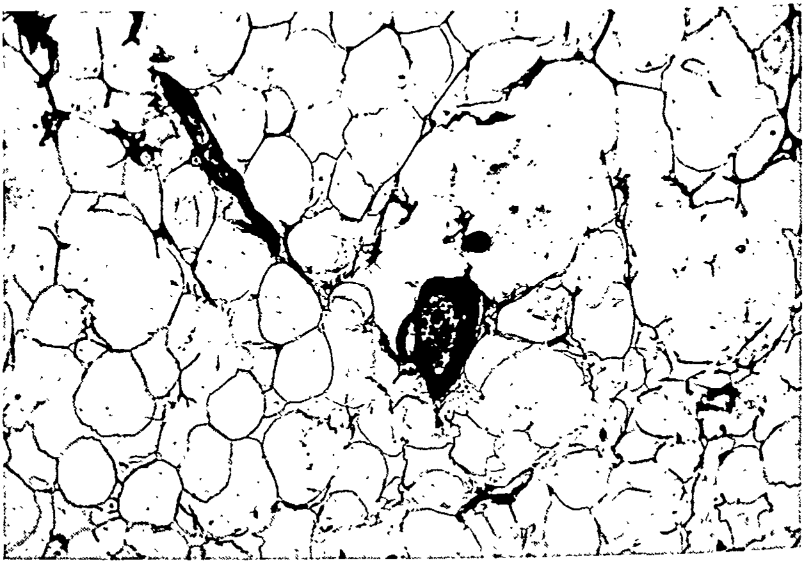

Fig. 6.—Microphotograph of the removed tumor. Typical fat cells are apparent without evidence of malignant change.

There are several reports, also, of the incidental finding at necropsy, of benign tumor of the heart, which had been, clinically, completely or almost completely silent. Such cases were reported by Arneson[8] in 1925 and by Guldberg[9] in 1928. In both instances, the tumor was a fibroma in the atrium. As a rule, malignant cardiac tumors run a comparatively brief course, and the outcome is invariably fatal.

The general feeling of the futility of treating primary tumors of the heart is not borne out by experiences during recent years, with the roentgenologic and operative treatment of these lesions. In 1937, Popp[10] reported a case of sarcoma of the right atrium, in which the symptoms improved, following x-ray radiation therapy instituted for the purpose of diagnosis. In 1940, Shelburne and Aronson[11] described a case of metastatic myeloblastoma in the heart, which was diagnosed by heart block and pericardial exudate, containing tumor cells. Following x-ray radiation therapy, the roentgenologic picture returned to normal, the heart block became partial on the electrocardiographic examination, and the patient was symptom free for many months, until metastases appeared in other organs. In 1940, Hsiung, Fzutu, Hsiehn and Lien, as quoted by Leubschitz, Lundsteen and Forchhammer,[6] also described the good effects of roentgen treatment in a metastatic cardiac tumor, which had arisen from a primary reticular sarcoma of the nose. In this particular instance, the roentgen picture, which had shown an enlarged heart shadow with kymographic signs of exudate, became normal following the roentgen treatment. The extremely low mortality rate attending exploratory thoracotomy, the availability of the modern antibiotics and the intriguing possibilities of intracardiac surgery, which might become possible following the clinical use of extracorporeal heart and lung machines now on the horizon, should make possible, in the near future, the surgical removal of a very large number of benign tumors, such as myxomata.

Myxomas comprise the bulk (50 per cent) of the benign tumors commonly encountered in the heart. They are most usually pedunculated, and they arise from the endocardial surfaces of the auricles, so that they would be particularly amenable to surgical removal.

## SUMMARY AND CONCLUSIONS

1. A successful surgical removal of a primary lipoma of the heart is reported. As nearly as can be determined, this is the first report of a successful removal of a primary lipoma of the heart and the second instance of a successful removal of a tumor of the heart, including all varieties.
2. The tumor in the case presented is the largest primary one, malignant or benign, which has been found reported in the literature to date.
3. Primary lipoma of the heart is a pathologic rarity, only two proved cases having been reported previously.

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# SURGICAL TREATMENT OF EPIPHRENIC DIVERTICULUM OF THE ESOPHAGUS

## REVIEW OF LITERATURE AND REPORT OF CASE TREATED BY RESECTION AND ESOPHAGOGASTROSTOMY

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DIVERTICULA of the lower portion of the esophagus are relatively uncommon, whereas those located in the terminal portion just above the diaphragm, termed supradiaphragmatic or epiphrenic diverticula,[38] are even less frequently encountered. In a review of the literature up to 1933, Granet was able to collect forty-nine cases, only thirty-one of which he accepted as proved cases of epiphrenic diverticula. Vinson, who reviewed the records of the Mayo Clinic up to 1934, discovered forty-two cases of diverticula of the thoracic esophagus, twenty-four of which were located in the lower third; less than half of these patients were considered to have symptoms that could be definitely associated with the diverticulum, a fact which indicates another reason why they are infrequently encountered as clinical problems. In an analysis of 1,600 patients admitted to the Outpatient Department of Massachusetts General Hospital because of dysphagia, Macmillan found less than one per cent due to diverticula of the thoracic esophagus. Further evidence of the rarity of this form of esophageal diverticulum is found in its relative incidence in comparison with the pharyngoesophageal type. Thus, Harrington encountered only eight cases of the epiphrenic type during the same period that he treated surgically 216 patients with pharyngoesophageal diverticula, and Lahey[26] reported a somewhat similar experience with six cases of the former to 211 of the latter type.

The paucity of the literature on the surgical management of epiphrenic diverticula of the esophagus is a further reflection of their infrequent occurrence, at least as clinical problems. Even as late as 1949 Poppe and Berg, in a report of a case of a patient successfully treated by diverticulogastric anastomosis, stated that in their review of the literature they had been able to find no previous report of the successful application of this method of treatment and only one case of successful resection. Although a more careful review of the literature has disclosed a number of previously recorded cases in which both of these methods of treatment had been successfully applied, there is nonetheless a dearth of reports on the surgical management of this type of esophageal diverticulum. For these reasons, and because no report has been found of the clinical application of the technical procedure used in the case to be described, a brief review of the surgical considerations of epiphrenic diverticula of the esophagus seems desirable.

These diverticula are generally considered to be of the pulsion type resulting from herniation of the mucous membrane through the muscular wall, probably in an area of congenital weakness, although the etiology is still ob-

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scure. The opening of the pouch is usually located in the posterior wall, more often directed toward the right than the left side, about 5 to 10 cm. above the cardia. The wall is composed of mucosa, submucosa, muscularis mucosa, and an outer fibrous layer of varying density. Infiltration of the wall of the sac with inflammatory cells is common, and the mucosa may become ulcerated. As the sacculation increases in size, it tends to assume a more dependent position in relation to the esophagus with distortion, angulation, and partial obstruction of the esophagus distal to the opening of the pouch. Accordingly, food passing down the esophagus tends to be shunted into the pouch. Cardiospasm has been considered a frequently associated factor and even of etiologic significance by some,[7, 19, 33, 35, 37, 44, 49] but we, like others,[2, 20, 21, 34] doubt this relationship, since diverticula are rarely found in patients with cardiospasm or achalasia. Probably the commonest complications are inflammation of the wall of the diverticulum, ulceration, and associated esophagitis, but perforation, hemorrhage, and malignancy have occasionally been encountered. Stricture of the esophagus distal to the diverticulum, which in the case to be described was largely responsible for the severity of symptoms, is not an unlikely complication in view of the progressive distortion and obstruction of the esophagus by the diverticula and the consequent prolonged esophagitis and spreading chronic inflammatory reaction.

Until rather recently most authors writing on this subject advocated conservative treatment because most patients had few or no symptoms, and operation was associated with considerable risk. It is now generally agreed, however, that surgical treatment is the method of choice and should be considered in all esophageal diverticula-producing symptoms. The surgical procedures that have been employed in the treatment of epiphrenic diverticula may be classified into four categories: (1) anastomosis between the sac and the stomach, (2) extirpation of the sac, (3) diverticulopexy, and (4) inversion of the sac. Historically, anastomosis between the diverticulum and the stomach was the procedure advocated, having been proposed in 1908 by Lotheissen, although Gosset, in 1903, had suggested this procedure for strictures of the cardia and had demonstrated its feasibility in cadavers and dogs, using a transpleural approach. In a subsequent publication Lotheissen[29] referred to a case in which this procedure was done using an abdominal transdiaphragmatic approach. Other German surgeons[15, 17, 22, 41] have employed this type of operation with apparently good results, while Henschen has been credited with the report of the first successful case in which this operation was done from above the diaphragm. Sauerbruch[41] advocated performance of the procedure in two stages, the first consisting of thoracotomy with mobilization of a portion of the fundus of the stomach through an opening in the diaphragm and suturing it to the diverticulum, and the second stage, done four weeks later, to complete the anastomosis. More recently two reports of its successful application have appeared in the American medical literature.[31, 35]

Extirpation of the diverticulum was first performed by Enderlen, in 1910, but the patient succumbed; Stierlein had a similar experience in 1916. Clairmont, in 1924, is believed to have performed the first successful extirpation using an abdominal transdiaphragmatic approach. The year before, however, Sauerbruch,[39] in describing a new method of exposing the posterior mediastinum to reach the esophagus, which he termed transpulmonary exposure of the esophagus, reported a case of perforation of an esophageal diverticulum into the lower lobe of the right lung successfully managed by staged operations. Several years later Sauerbruch[40] reported three successful cases, using an anterior mediastinotomy approach in the first, posterior mediastinotomy in the second, and a transpleural approach in the third. This latter approach was used successfully by Meyer to extirpate an epiphrenic diverticulum, according to Torek in 1927. The transpleural approach for extirpation of the sac was also utilized successfully by Fick in 1929, and by Barrett in 1933, and the posterior mediastinotomy approach by Vos in the case reported by Quartero in 1931. More recently a number of surgeons[1, 5, 8, 11, 16, 20, 26, 43, 50] have reported favorably on their experience with this method, the procedure consisting essentially in a transpleural approach through the right or left side, depending on the direction of the pouch, careful mobilization of the sac to expose its neck, complete excision, and repair of the opening in the esophagus by a two- or three-layer anastomosis with closure of the overlying mediastinal pleura.

In an attempt to avoid the development of mediastinitis and empyema, which were the complications feared by the early efforts to extirpate the sac, Lahey,[24] following the suggestion of others[18, 23, 27, 42] for pharyngeal diverticula, advocated and employed diverticulopexy. In this procedure the sac is carefully freed from its surrounding attachments and mobilized upward, and the fundus of the sac is fixed with interrupted sutures to the pleura in the paravertebral gutter so as to afford dependent drainage.

Another form of surgical treatment designed to avoid infection from opening the sac consists in inversion of the diverticulum. This procedure was first employed for pharyngeal diverticula by Girard in 1896. It was also advocated for thoracic esophageal diverticula by Torek, in 1927, who considered it the safest method. It was done by Turner[47, 48] through a posterior extrapleural approach, but the pleura was accidentally injured and the patient subsequently died of pneumonia, empyema, and a bronchopleural fistula.

From an analysis of these various methods for the surgical treatment of epiphrenic diverticula, it would seem that complete extirpation with proper repair of the opening in the esophagus is the method of choice, since it usually achieves the ideal objective, that is, removal of the lesion and restoration of normal anatomy and function. This objective, however, cannot always be achieved by this method, owing to certain pathologic changes consequent to long-standing ulceration, esophagitis, and stricture formation which may extend beyond the neck of the diverticulum to involve the segment of the esophagus just distal to the pouch. Such a condition was encountered in the case to be described herein. Under these circumstances it would appear that resection of the lower portion of the esophagus including the diverticulum and cardia of the stomach followed by esophagogastrostomy provides a practical method for dealing with this condition. Experience with this technique in the treatment of other lesions of the esophagus has demonstrated its safety and efficacy. Since no reports of the clinical use of this form of surgical treatment for epiphrenic diverticula have appeared in the literature, it seems desirable to direct attention to its employment under these circumstances by presentation of the following case.

## REPORT OF CASE

J. B. T., a white man, aged 59 years, was first seen on Sept. 13, 1949, with a history of difficulty in swallowing of nine years' duration. Ingested liquids and solid foods would "hang up" beneath the lower end of the sternum, and occasionally he would vomit an hour or two after meals. The regurgitated food was foul smelling and extremely distasteful. He was frequently awakened at night by a "scratching" sensation in his throat which provoked bouts of coughing. Soon after the onset of these symptoms he consulted a physician who, after barium study of the esophagus, made a diagnosis of diverticulum of the lower portion of the esophagus. This condition was treated conservatively with moderate relief of symptoms; however, during the past year the symptoms had increased in severity and serial roentgenograms showed the diverticulum to be enlarging.

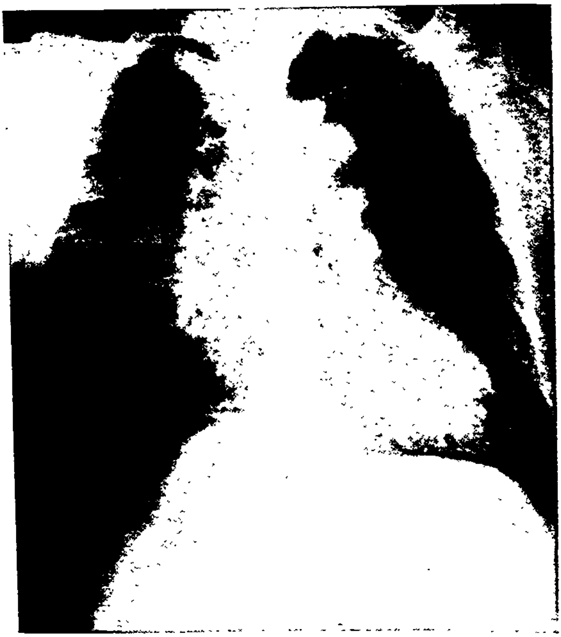

Fig. 1.—Oblique roentgenogram of the esophagus following ingestion of barium demonstrating the diverticulum with dilatation and coiling of the esophagus above and narrowing below.

Examination revealed a well-nourished and well-developed white man with no gross abnormalities. An esophagogram disclosed a large diverticulum of the distal end of the esophagus arising from the left lateral wall just above the cardiac end of the stomach. Above the diverticulum the esophagus was quite dilated and somewhat tortuous, whereas distal to it a definite narrowing could be demonstrated (Fig. 1). A diagnosis was made of epiphrenic diverticulum of the esophagus with stricture formation, and the patient was admitted to the hospital for surgical therapy.

On Sept. 15, 1949, under endotracheal nitrous oxide and ether anesthesia, with the patient in the right lateral decubitus position, the eighth rib was resected subperiosteally.

and through its bed the left thoracic cavity was entered. Preliminary exploration confirmed the roentgenographic observation of a diverticulum protruding from the left posterior lateral wall of the esophagus just anterior to the aorta and immediately above the cardia. The mediastinal pleura was incised, and the lower third of the esophagus, including the diverticulum, was mobilized. The diverticulum was found to measure approximately 5 cm. in length and 3 cm. in diameter. The esophagus distal to the opening of the diverticulum and extending to the cardia appeared markedly constricted, with a firm thickened wall moderately adherent to the surrounding tissue. In the presence of this rather pronounced degree of chronic esophagitis and stricture formation, it was believed that diverticulectomy alone might not provide adequate relief of the obstructive symptoms. For this reason it was considered preferable to resect the segment of esophagus involved in the chronic inflammatory process along with the diverticulum from just above the opening of the pouch to the cardia and restore continuity by esophagogastric anastomosis. The diaphragm was divided from the esophageal hiatus anterolaterally for a distance of 8 cm. Through this incision the fundus and cardiac end of the stomach were mobilized by division of the vasa brevia and left gastric vessels. After approximately the upper fourth of the stomach had been mobilized, it could be brought up into the chest without difficulty. The stomach was divided at the cardia between Payr's clamps, and the gastric stump closed with a running suture of 000 chromic catgut followed by a row of interrupted Lembert sutures of cotton (Fig. 2). The fundus of the stomach was pulled up into the mediastinum, and esophagogastrostomy was performed utilizing a two-layer anastomosis with cotton (Fig. 3). The stoma was constructed in the share of an L to increase its diameter.[6] The fundus of the stomach was

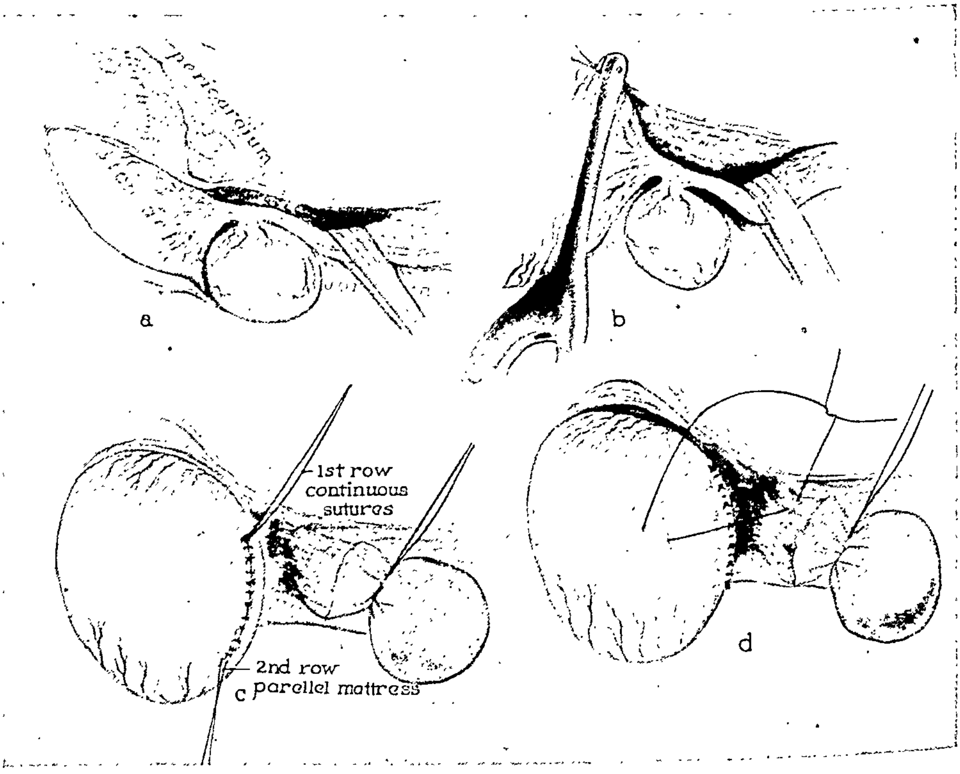

Fig. 2.—Drawing made from authors' case showing (*a*) mobilization of lower esophagus, diverticulum, cardia, and fundus of stomach after opening diaphragm, (*b*) division of stomach just below cardia, (*c*) closure of the gastric stump, and (*d*) beginning esophagogastric anastomosis.

anchored to the mediastinal pleura and the diaphragm closed about the stomach. A No. 32 Pezzar catheter was placed in the ninth interspace in the posterior axillary line for underwater drainage, and the thoracic wound was closed in layers with cotton sutures. During the procedure, the patient received 1500 c.c. of blood, and left the operating room in good condition.

Examination of the gross specimen revealed an esophageal diverticulum of the pulsion type. Approximately 0.5 cm. distal to the mouth of the diverticulum the esophagus was constricted to an internal diameter of 0.5 cm. with a firm, thickened, and somewhat scarred wall. Histologic examination revealed thickened stratified squamous epithelium lining the diverticulum, but no evidence of anaplasia. The remainder of the mucosa for the most part was artifactually absent; however, at one end there was glandular mucosa representing the cardia of the stomach. The smooth muscle layers were well developed in the wall of the esophagus, but were entirely absent in the wall of the diverticulum. Mild focal lymphocytic infiltration was seen throughout the layers of the esophagus. The pathologic diagnosis was pulsion type diverticulum of the esophagus.

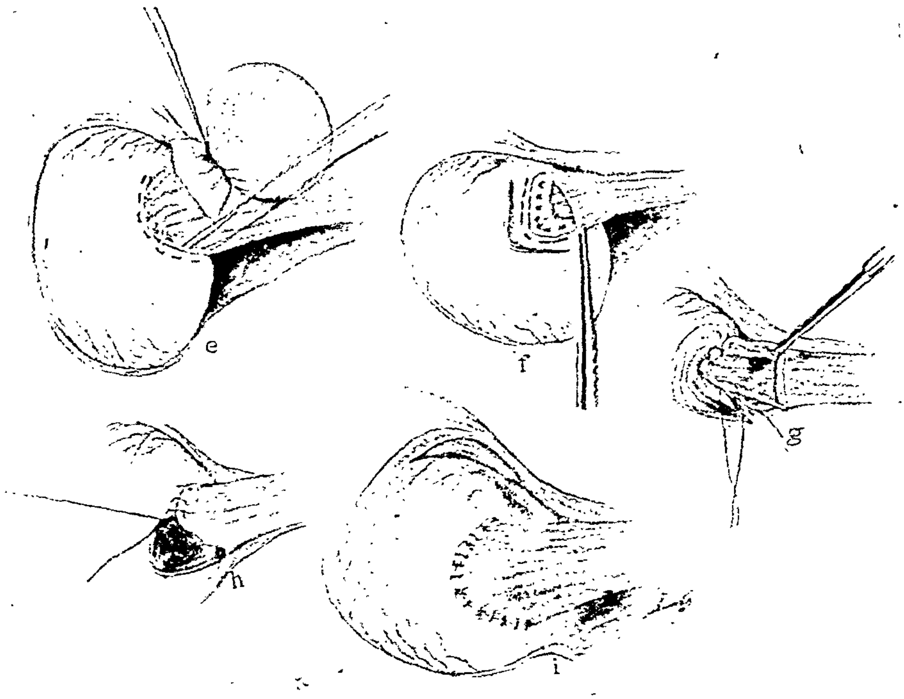

Fig. 3.—Drawing showing steps in technique of esophagogastrostomy performed on authors' case continued from Fig. 2. (*e*) The first posterior row of sutures is placed between the posterior wall of the esophagus and the anterior wall of the fundus of the stomach with an extension upward for about 2 cm. along the left lateral margin of the esophagus. (*f*) The esophagus and stomach are opened by incisions just distal to this first row of sutures, producing L-shaped openings to increase the size of the stoma. (*g*) Placement of inner row of posterior interrupted sutures of quilting cotton, and (*h*) first layer of anterior sutures to provide careful approximation of mucosal edges, care being taken to place sutures so that knots are within the lumen as suggested by Churchill and Sweet. (*i*) Completion of anastomosis by outer layer of anterior sutures.

The postoperative course was uneventful. The drainage catheter was removed on the third postoperative day, and small oral feedings were begun the fifth postoperative day. The diet was gradually increased, and the patient was discharged ten days after operation.

Following his return home he was able to eat without difficulty, and when examined approximately one and one-half years after resection of the diverticulum the patient was asymptomatic, and barium study of the esophagus at that time revealed an adequate and well-functioning esophagogastric stoma (Fig. 4). When last observed on Sept. 11, 1951, approximately two years after operation, he was still well and asymptomatic.

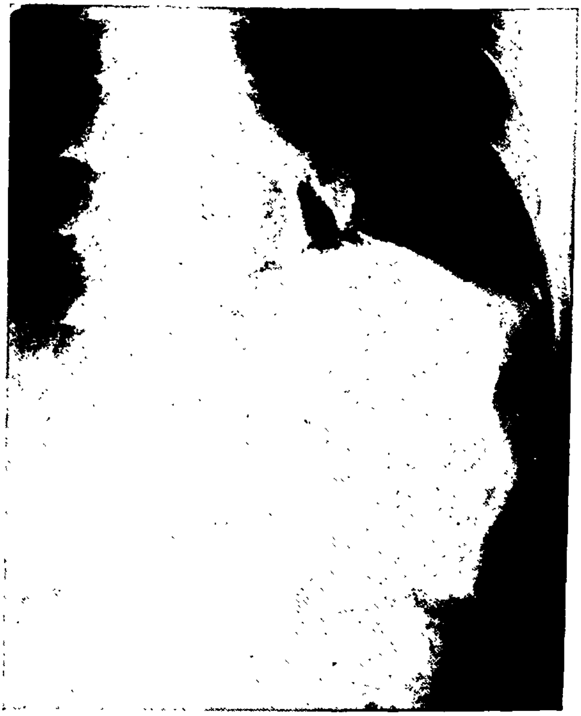

Fig. 4.—Barium study one year after operation showing a normally functioning esophagogastric stoma.

## SUMMARY

1. Diverticula of the lower esophagus, particularly those located in the terminal portion and termed supradiaphragmatic or epiphrenic diverticula, are infrequently encountered. Although the pathogenesis of these diverticula has not been clearly established, they are generally considered to be of the pulsion type resulting from herniation of the mucous membrane through the muscular wall, probably in an area of congenital weakness. Symptoms are produced by the occurrence of certain complications, such as partial obstruction resulting from distortion and angulation of the esophagus or inflammation and ulceration with consequent stricture formation as in the case described, or from perforation, hemorrhage, or malignancy.

2. Surgical treatment is the method of choice and should be considered in all esophageal diverticula-producing symptoms. From a review of the literature the procedures that have been advocated and employed in the surgical treatment of epiphrenic diverticula may be classified into the following categories: (1) anastomosis between the sac and the stomach, (2) extirpation of the sac, (3) diverticulopexy, and (4) inversion of the pouch.

3. Complete extirpation of the diverticulum with proper repair of the opening in the esophagus is considered the method of choice. In the case reported this procedure was not considered satisfactory, owing to the presence of marked stricture formation of the esophagus distal to the diverticulum. For this reason, the procedure employed consisted in resection of the lower esophagus including the diverticulum and cardia of the stomach followed by esophagogastrostomy. Although no report has been found of the use of this procedure for this purpose, in light of this experience it is recommended as a practical method for dealing with this condition.

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# MISINTERPRETATION OF FINDINGS IN CATHETERIZATION OF ANOMALOUS PULMONARY VEINS ADJACENT TO A MEDIASTINAL DERMOID CYST

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THE diagnosis of a mediastinal dermoid cyst is not always difficult when there is evidence of a progressively enlarging tumor mass in the anterior mediastinum in an individual in the third decade.[1, 2] The recognition of anomalous pulmonary venous drainage, however, depends upon special procedures, since there are no reliable physical signs by external examination of the chest.[3] These procedures include direct inspection of the hilar vessels at either the operating table[4] or the post-mortem table,[5] and demonstration by angiocardiography or by the fortuitous intubation of one or more such veins during venous catheterization of the heart.[3, 6, 8-11] Because the latter approach to a diagnostic problem provided information which was misinterpreted prior to exploratory thoracotomy, the following case is presented:

C. M. J., a 23-year-old white Naval seaman, was referred for diagnostic studies for possible congenital cardiovascular disease. A routine chest roentgenogram one month previously showed an enlarged cardiac shadow (Fig. 1), which was not apparent on a film taken five years earlier (Fig. 2). The possibility of a pericardial cyst was considered also. The patient had been asymptomatic except for occasional palpitations on exertion. He was 6 feet, 3 inches tall, weighed 195 lbs., and appeared to be in excellent health. There was no evidence of cyanosis, clubbing, venous distention, or edema. A slight precordial bulge was present, but there was no marked intercostal pulsation and no thrill. On percussion the area of cardiac dullness extended from the left midclavicular line to a point 4 cm. lateral to the right sternal margin in the third and fourth interspaces. The second pulmonic sound was accentuated, and there was a grade 2 systolic murmur along the left sternal border. There was no diastolic murmur. There was a sinus bradycardia with a rate of 58 per minute; the blood pressure was 135/80 mm. Hg. The remainder of the physical examination was normal.

Chest fluoroscopy, cinefluorography, and radiography revealed clear lung fields with apparently normal bronchovascular markings (Fig. 1). The transverse diameter of the heart was widened, however, due to a pulsating shadow which extended to the right and anteriorly. It was continuous with the cardiac shadow and was observed to diminish in size with the Valsalva maneuver (Fig. 3). In the right anterior oblique view, it extended anteriorly toward the rib cage, thereby simulating an enlarged right heart.

An electrocardiogram showed an intermediate position, or normal axis deviation, and sinus bradycardia of 55 per minute. There was no evidence of a conduction defect or ventricular hypertrophy, and the tracings were within normal limits. The hemoglobin concentration was 15.1 Gm. per cent (Van Slyke determination). The urine was normal. An exercise tolerance test using a 10-minute step-test technique[12] was entirely normal; the physical

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TABLE I

| LOCATION OF CATHETER TIP | $O_2$ SATURATION (%) | PRESSURES (MM. HG) |
|---|---|---|
| Superior vena cava | 73 | -- |
| Inferior vena cava | 82 | -- |
| Right auricle | 77 | 7-10* |
| Region of the mass | 93 | 22-25* |
| Right ventricle | -- | 30/2-5 |
| Pulmonary artery | 79 | 26/15 |
| Femoral artery | 96 | -- |

*Variation in mean pressure with respiration.

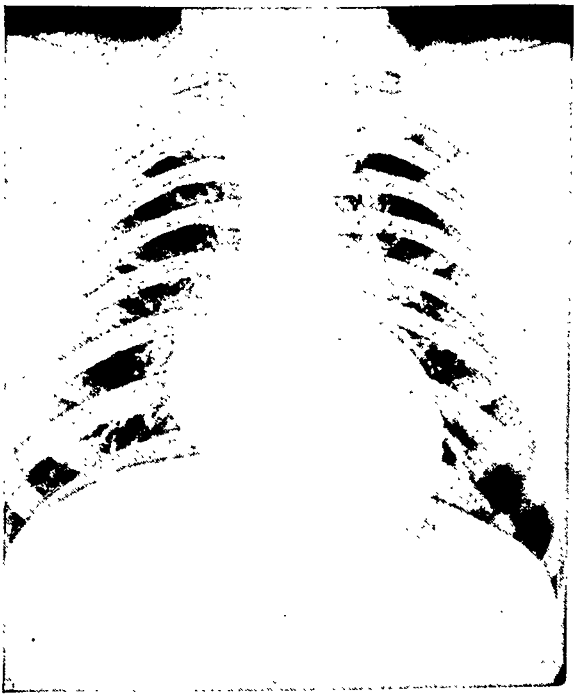

Fig. 1.—Chest roentgenogram of C. M. J. taken in December, 1950. Note the unusual contour of the right border of the cardiac shadow.

fitness index was 22.8. There were no abnormalities in the chest-lead electrocardiogram during the exercise. Venous catheterization of the right heart yielded the information noted in Table I.

Since the oxygen consumption was 320 ml. per minute, and the AV oxygen difference was 37 ml. per liter, the cardiac output at rest was a high normal value of 8.65 liters per minute. The stroke volume was 129 milliliters. Total pulmonary resistance was estimated to be about 157 dynes sec. cm.$^{-5}$, whereas that of the periphery was about 906 dynes sec. cm.$^{-5}$. The tip of the catheter was inserted into an anomalous vessel entering the superior vena cava (Fig. 4) where highly oxygenated blood was withdrawn. The mean pressure at this point (22 to 25 mm. Hg) was higher than that observed for either the right auricle (7 to 10 mm. Hg) or the left pulmonary artery (14 to 17 mm. Hg), and rose to 60 mm. with performance of the Valsalva test. A somewhat damped arterial pressure pattern was recorded during the Müller test, with a mean pressure of around 30 mm. Hg. Accordingly this vessel was considered to be a communication with one of the systemic arteries. With moderate leg exercises, the pressure in the left pulmonary artery rose to 36/16 mm. Hg.

The observations reported above confirmed the findings of the referring physician that the patient had an abnormal configuration of the heart by roentgenogram examination. The possibility of a pericardial cyst seemed unlikely because of the rather striking reduction in size with the Valsalva maneuver, which was attributed to the effects of greater intrathoracic pressure on venous return to the heart. Insertion of the catheter tip into the region of the

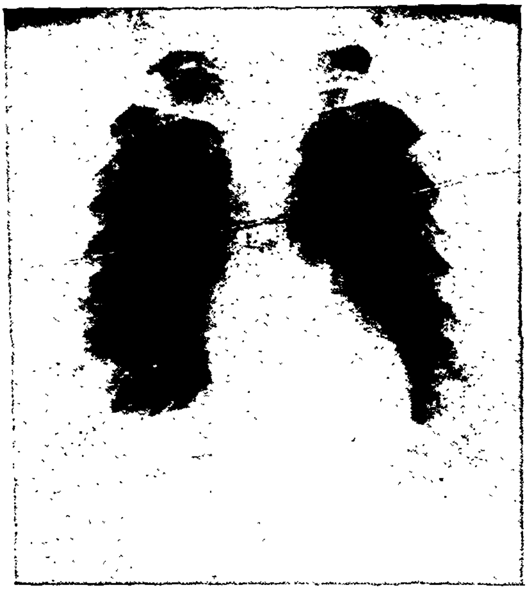

Fig. 2.—Chest roentgenogram taken in July, 1945. Despite the slight convexity of the right auricular shadow, it was considered to be normal.

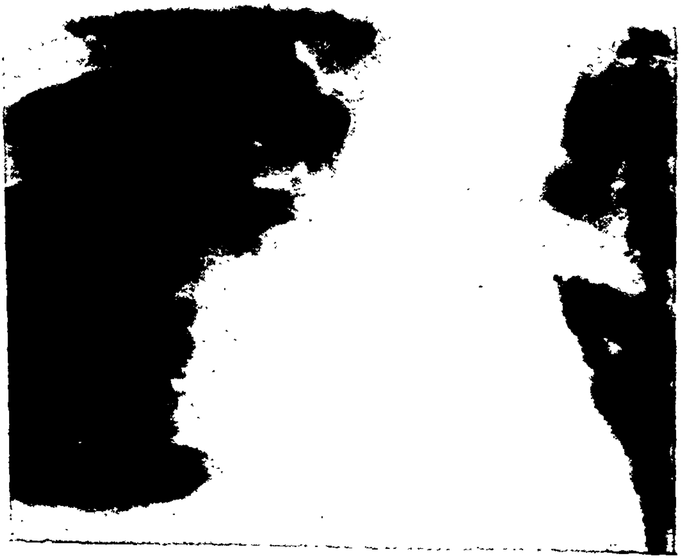

Fig. 3.—Spot film of the right side of the heart taken during cardiac catheterization to demonstrate the reduction in the size of the mass during the Valsalva maneuver. (Compare with Fig. 4.)

mass during venous catheterization of the heart provided three observations of interest: there was an anomalous vessel entering the right auricle, it contained blood that was highly oxygenated, and the blood pressure was appreciably higher than that of the right auricle, increasing to 60 mm. of mercury with the Valsalva maneuver. The pressure pulse which was recorded graphically resembled an arterial pattern. Although the diagnosis was uncertain, the combined findings of physical and roentgenogram examinations, together with the cardiac catheterization indicated an arterioauricular aneurysm or an angiomatous tumor with an auricular communication. Because of the appearance and rapid enlargement of this mass within a five-year period (see Figs. 1 and 2), surgical exploration was considered mandatory.

A right exploratory thoracotomy was carried out on March 22, 1951. Upon entering the chest, a large dermoid cyst was identified. It was attached to the pericardium over the right auricle and the ascending aorta by a broad pedicle. The mass (Fig. 5) was removed without untoward incident (Fig. 6). In order to explain the cardiac catheterization findings, a dissection of the pulmonary hilus was undertaken. The pulmonary venous drainage of the right lung consisted of three veins. A single large vein draining the right upper lobe emptied directly into the superior vena cava approximately 8 cm. above the pericardial reflexion. A smaller vein from the right middle lobe emptied anteriorly and directly into the right auricle. The inferior pulmonary vein was noted to be in its normal position. No further attempt to trace the course of this latter vein was made.

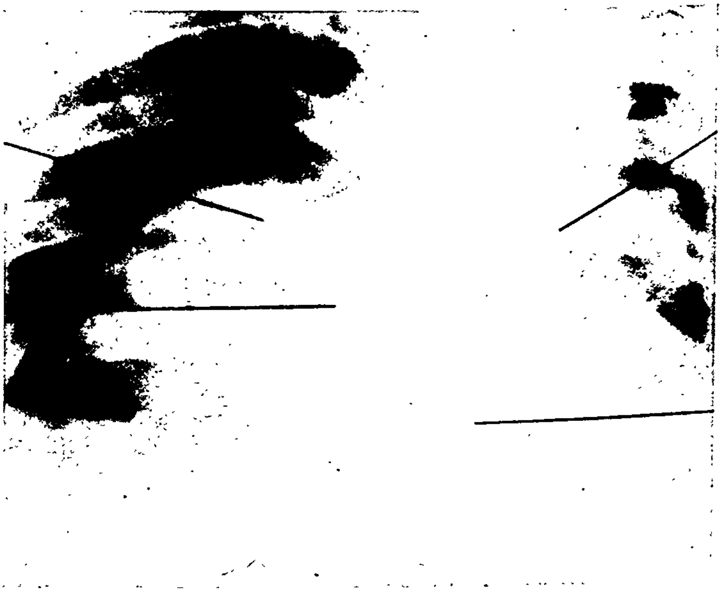

Fig. 4.—Spot film of the heart taken during cardiac catheterization. Note the tip of the catheter in the region of the mass. Oxygen saturations and blood pressures in mm. Hg as indicated.

The patient's postoperative convalescence was uneventful. He was discharged on the twelfth postoperative day completely ambulatory and without complaint. He has since returned to full-time work.

## DISCUSSION

Had not cardiac catheterization been performed on this patient, the presumptive diagnosis would have been dermoid or pericardial cyst. The history of palpitations and the fluoroscopic evidence of a pulsating mass, as observed in this patient, have been described in previous cases of dermoid cysts. Venous pulsations and variations in density with either the Valsalva or Müller experiments have not been striking by roentgenoscopic examination in patients with anomalous pulmonary veins.[7] The preoperative venous catheterization of the heart in this patient, however, provided information which was misinterpreted.

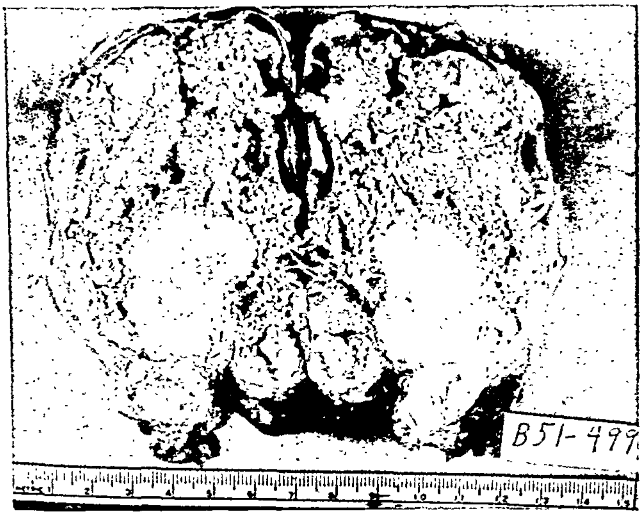

Fig. 5.—The mass measured 7 × 11 cm. The outer surface was golden yellow and somewhat nodular. On section a cheesy material was present intermingled with hair. Microscopic examination revealed only ectodermal elements. The final diagnosis was dermoid cyst.

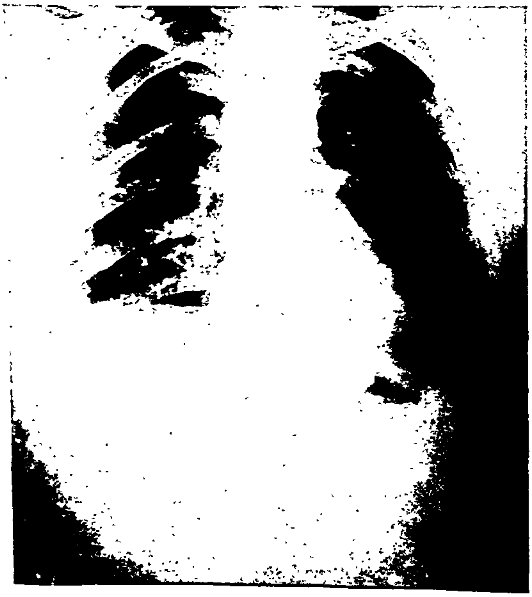

Fig. 6.—Chest roentgenogram taken ten days after removal of the dermoid cyst. The vascular markings below the right hilus are more conspicuous since the overlying tumor has been removed.

This was due to the fortuitous insertion of the catheter tip into one of the anomalous pulmonary veins, the withdrawal of highly oxygenated blood, and observing an appreciably higher pressure than in the adjacent right auricle. Possibly a lateral radiograph would have been helpful in localizing the position of the catheter in relation to the mass, but even this could have been misleading because of the fact that the mass was attached to a pedicle.

Although nearly 300 cases of mediastinal dermoid cyst have been reported,[1, 2] apparently none have been associated with anomalous pulmonary venous drainage. In this patient, the venous anomaly was not recognized until the hilar structures had been dissected carefully at the operating table. Anatomists have found the incidence of anomalous pulmonary venous drainage to be about 0.7 per cent.[13] This patient represents the fourth example of this anomaly that the authors have encountered within the past year. Hence these findings suggest that it is more common than usually stated. Unless pathologists are alerted to the possibility, it is likely that the exact pathology will be overlooked in the type of autopsy routinely performed.

When less than 50 per cent of the pulmonary venous return enters the right auricle the patient has no symptoms,[3, 6] there is little likelihood of decompensation, and the life span need not be shortened.[5] With total venous drainage of the lungs into the right heart, there must be either a septal defect or patency of the ductus arteriosus in order to supply the major circulation with blood that has been oxygenated. In these cases not only is the right ventricle hypertrophied, but the left heart is smaller than usual.[5] Closure of the ductus arteriosus diminishes the blood flow to the greater circulation, leads to dilatation and failure of the right heart, and accounts for the death of the patient in early infancy.[5] With only partial anomalous venous drainage, either collapse or removal of part or all of the lung with normal venous drainage is dangerous.[3, 4, 6] Even atelectasis of the normal lung may be hazardous to the patient.[6] It has been suggested that some of the immediate accidents following pneumothorax may be attributed to such a mechanism.[3] Under any of these circumstances the reduction in the amount of oxygenated blood available to the greater circulation becomes critical.

## SUMMARY

1. A report is made of a patient who, primarily on the basis of cardiac catheterization studies, was regarded clinically as having an arterio-auricular aneurysm or an angiomatous tumor with an auricular communication. However, upon surgical exploration he was found to have a dermoid cyst in the mediastinum and anomalous veins going from the right lung to the superior vena cava and right auricle.

2. The danger of any surgical therapy of the lung with normal venous drainage, in the presence of anomalous venous drainage of the opposite lung, is emphasized again.

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# CASE REPORT—AN UNUSUAL MEDIASTINAL CYST

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THIS case of a mediastinal cyst is presented because of the difficulties experienced in diagnosis together with an unusual response to certain therapeutic agents.

The patient, Mr. S., a 54-year-old white man, was apparently in good health until February, 1948, when he suddenly lost his voice, being unable to speak above a whisper. Approximately one week later he developed a mildly productive cough with mucoid sputum, once blood-streaked. Approximately two weeks later he noted a numbness and stiffness of the fourth and fifth fingers of his left hand, but this symptom gradually disappeared within a few days. There were no associated weight loss, dyspnea, fever, or difficulty in swallowing. The past history was noncontributory, except for treatment for syphilis in 1931 and again in 1941.

When first seen in March, 1948, the physical examination was negative except for hypertension (200/98) and a complete paralysis of the left true vocal cord. Laboratory studies, including a blood Kahn test, were all within normal limits. An upright posteroanterior and left lateral film of the chest revealed a hazy shadow of uniform density along the right superior mediastinum and extending out to the left for a distance of 2.5 cm. from the lateral border of the spine near the aortic knob. The lateral border of this opacity had a smooth contour. Esophagograms revealed a compression of the esophagus in the region of the fourth and fifth thoracic vertebrae, with a slight displacement of the upper esophagus and trachea toward the right just within the thoracic inlet. Esophagoscopic examination revealed a slight compression of the left wall of the esophagus in the upper third. The esophageal mucosa appeared smooth and normal throughout with no stenosis or intrinsic tumor visualized. Bronchoscopic examination showed the trachea to be compressed on its left lateral surface with a shift toward the right in the same location. The remainder of the bronchial tree was normal. A bronchial washing of the left main bronchus was reported by the pathologist to be negative for tumor cells.

As a result of the above findings, we considered the diagnosis in this case to be a bronchiogenic carcinoma with invasion of the left recurrent laryngeal nerve, and therefore not curable surgically. Since the patient's only incapacity at the time was his weak voice, it was our feeling that he should be treated conservatively for the present, and therefore we withheld Methyl-bis and x-ray therapy until needed. It was decided that an exploratory thoracotomy and biopsy would only cause the patient further discomfort and not add materially to the understanding of his case.

The patient continued to work until January, 1949, when he was admitted to the Baptist Memorial Hospital in Memphis because of severe dyspnea, cyanosis, fever, and peripheral circulatory collapse. Physical examination revealed a fever of 102° F., a pulse rate of 120 per minute, respiratory rate of 26 per minute, a complete paralysis of the left vocal cord, and moist râles throughout both lungs. X-ray examination of the chest (Fig. 1) showed no apparent increase in the mediastinal mass. There was, however, a marked pneumonitis involving the bases of both lung fields. Treatment consisted of nasal oxygen, intravenous fluids, sedation, and penicillin intramuscularly. The respiratory difficulty gradually subsided, and within approximately ten days he was afebrile and able to do without oxygen. On Jan. 26, 1949, Methyl-bis therapy was instituted, 10 mg. being given daily

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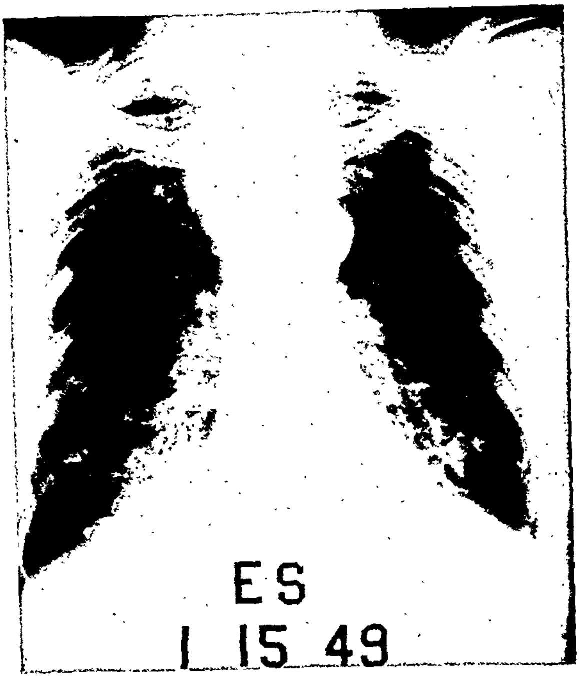

Fig. 1.—Patient E. S., Jan. 15, 1949. There is a hazy shadow of uniform density along the right superior mediastinum which extends out to the left for a distance of 2.5 cm. from the lateral border of the spine near the aortic knob. The lateral border of this opacity has a smooth contour. There is also some pneumonitis involving the bases of both lung fields.

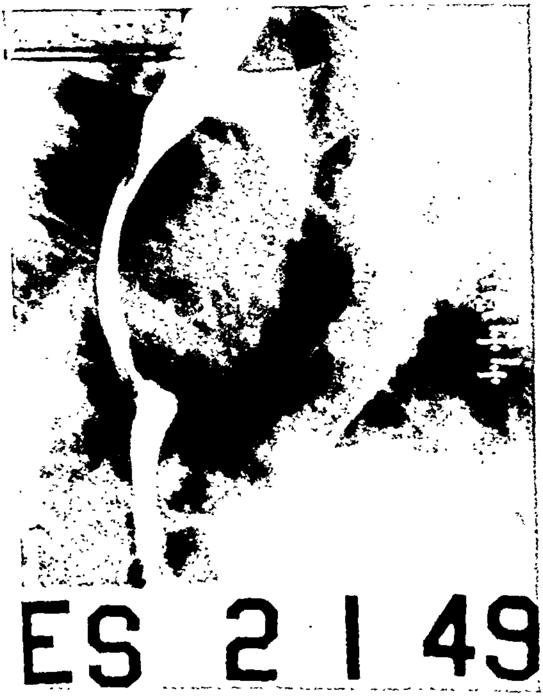

Fig. 2.—Esophagogram Feb. 1, 1949. This reveals a *smooth* compression of the esophagus in the region of the fourth and fifth thoracic vertebrae with slight displacement toward the right of the upper esophagus and trachea just within the thoracic inlet. The tumor is definitely between the trachea and the esophagus. The *smooth* contour of the mass suggested a benign neoplasm, but we discounted this possibility because of the presence of the paralyzed vocal cord and were thus led *astray for some time.*

intravenously for four consecutive days. Following this the patient showed considerable symptomatic improvement, exhibited by more comfortable breathing, and a lessening of his severe cough. A roentgenogram of the chest with barium in the esophagus on Feb. 1, 1949 (Fig. 2), showed the esophagus to be displaced backward, and the trachea displaced to the right and forward. A long fusiform mass with a smooth contour separated the trachea and esophagus. No irregularity was evident in the contour of the esophagus or trachea. A kidney-ureter-bladder film revealed no enlargement of the liver, spleen, or kidneys. Films of the lumbar spine, pelvis, and hips were reported as negative.

A repeat bronchoscopic examination was done on Feb. 7, 1949, which revealed the trachea to be displaced anteriorly. Considerable mucopurulent sputum was observed coming from the orifices of all lobes. A bronchoscopic sputum specimen was taken, and this was reported as being negative for tumor cells. A repeat posteroanterior film of the chest taken on Feb. 8, 1949, revealed a *sharp diminution in the size of the mediastinal mass* and a clearing of the pneumonitis in both bases. The patient was placed on active bronchial drainage routine with ammonium chloride and discharged home on Feb. 9, 1949, with instructions to return within ten days for an exploratory thoracotomy. This procedure was now deemed advisable since there was a good possibility the mass was benign, because of the smooth borders of the tumor, and because of the lack of invasion of contiguous structures (except for the left recurrent laryngeal nerve) for the period of time the patient had been under observation. Upon his return to the hospital on Feb. 21, 1949, a posteroanterior roentgenogram of the chest (Fig. 3) showed the mediastinal mass to have almost completely disappeared. Because of this dramatic response to Methyl-bis therapy it was now thought that this

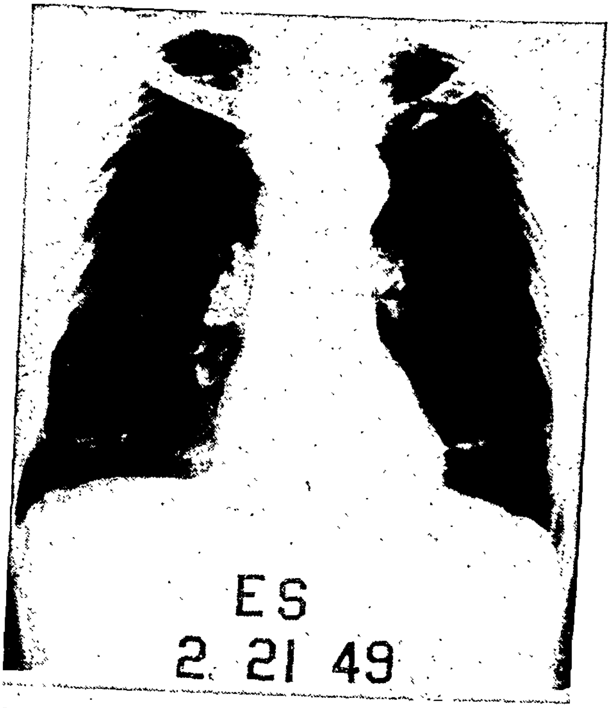

Fig. 3.—Feb. 21, 1949. After a course of Methyl-bis intravenously, 10 mg. once a day for four days, and intensive x-ray therapy to the superior mediastinum, there has been a marked decrease in the size of the tumor along the right superior mediastinum. This again confused us and made us believe that we were dealing with a bronchiogenic carcinoma or lymphoma as we have seen a similar response in this region in other cases.

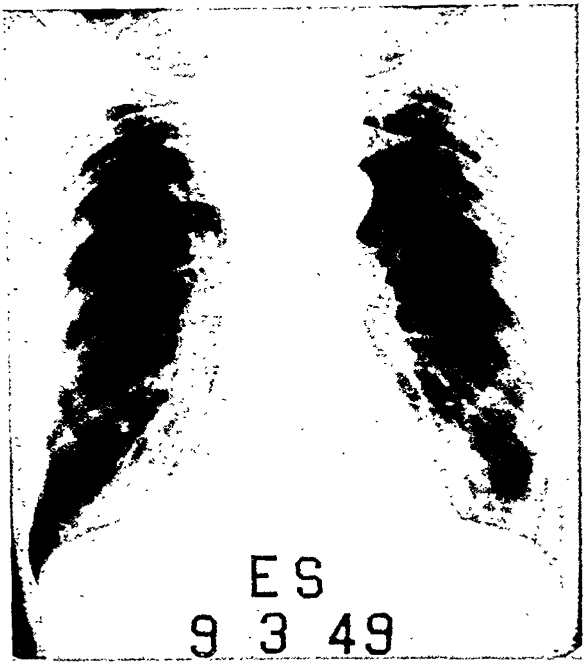

Fig. 4.—Sept. 3, 1949. The patient had fever and respiratory difficulty at this time and roentgenograms revealed an increase in the size of the neoplasm in the right upper chest, and again a pneumonitis in the right cardiophrenic angle. After the pneumonitis was controlled, an exploratory thoracotomy was decided upon, but was refused by the patient for some time.

On Oct. 1, 1949, operation was performed and a large tumor resected. This tumor occupied the triangle between the superior vena cava anteriorly, the azygos vein inferiorly, and the esophagus posteriorly. The cyst was removed in toto.

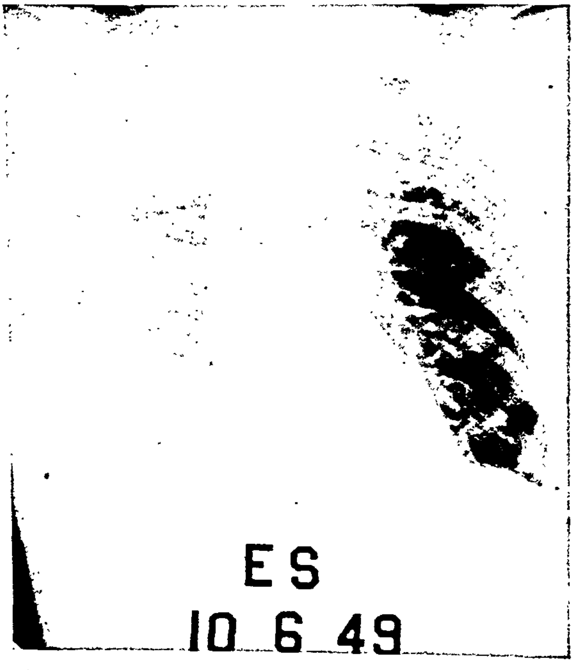

Fig. 5.—Oct. 6, 1949. Roentgenogram made a few days after operation reveals some pleural reaction over the entire right lung field but the tumor has been removed completely from the right superior mediastinum. The patient's convalescence since this time has been most gratifying and he is now back at full-time work.

was a case of lymphoma, either of the lymphosarcoma or Hodgkin's class, and not curable by surgery. Therefore the patient was again discharged, and instructed to return as an outpatient for x-ray therapy to the upper mediastinum.

The patient was admitted again to the hospital on April 15, 1949, with an acute respiratory infection. Temperature was 101° F., pulse rate was 104 per minute, and respiratory rate was 36 per minute. He exhibited signs of partial tracheal compression with associated bilateral decreased breath sounds. Posteroanterior roentgenogram of the chest showed the mediastinal mass to have resumed its former size and contour. The patient was again treated symptomatically and given 40 mg. of Methyl-bis in divided doses. One week from the date of admission he was feeling well, and was discharged home, ambulatory.

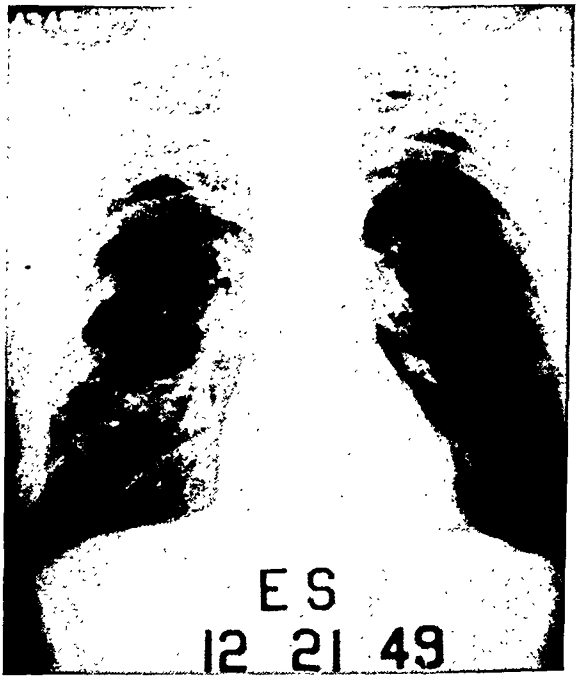

Fig. 6.—Follow-up roentgenogram, Dec. 21, 1949, shows marked clearing of the pleuritis on the right. The patient is asymptomatic except for hoarseness, and the paralyzed left *vocal cord is still unchanged.*

During May, 1949, the patient was given a second series of x-ray treatments to the upper mediastinum. However, he continued to have a severe productive cough with tenacious yellowish sputum and dyspnea, especially in the supine position. Since he received no immediate benefit from the second series of x-ray treatments, it was decided to try to keep the patient comfortable on Methyl-bis injections (10 mg.) intravenously every two weeks in the office, and to give blood transfusions when indicated. The patient continued to breathe comfortably and to work part time until Sept. 2, 1949, when he was admitted again to the hospital with extreme respiratory difficulty, fever, and a bilateral basal pneumonitis (Fig. 4). Treatment consisted of oxygen, aminophylline, and 5 per cent dextrose intravenously twice each day, postural drainage, and intramuscular and aerosol penicillin. The pneumonitis responded promptly and he was discharged home on Sept. 11, 1949.

On Sept. 28, 1949, the patient was readmitted to the hospital for a right exploratory thoracotomy, as our doubts grew as to his diagnosis. At operation a large cystic tumor, approximately 10 cm. in its longest diameter, occupied the triangle between the superior vena cava anteriorly, the azygos vein inferiorly, and the esophagus posteriorly. The cyst was removed in toto. Its contents were a thin crystal-clear watery secretion. The day following operation an emergency tracheotomy was necessary because of the paralyzed vocal cord, and edema of the vocal cords after endotracheal anesthesia. Otherwise, the postoperative course was uneventful. The tracheotomy tube was removed in a few days and the opening allowed to close by granulation. The patient was discharged home on Oct. 14, 1949, afebrile and asymptomatic, except for hoarseness (Fig. 5).

The cyst was reported by the pathologist to be a mesothelial-lined cyst compatible with a cavernous lymphangioma.

The patient was last seen on Aug. 7, 1950, at which time he was working full time with no symptoms referable to his chest.

## SUMMARY

A benign mediastinal cyst resembled clinically a bronchiogenic carcinoma with mediastinal invasion. The cyst decreased remarkably in size with x-ray therapy and Methyl-bis injections especially the latter. This decrease in size could be attributed to the vomiting and dehydration caused by the nitrogen mustard, or else due to shrinkage of the lymphatics that fill the cyst due to radiation therapy.

Having learned a lesson from his case, we now believe that exploratory thoracotomy is indicated for the patient with supposed bronchiogenic carcinoma with a paralyzed vocal cord, unless definite evidence of tumor exists elsewhere.

# AN ACCESSORY LUNG

## REPORT OF A CASE

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ANOMALIES of the tracheobronchial tree are numerous. However, there have been few reports in the literature of a true accessory lung. The so-called accessory lung can be classified into two main types: the lung generally stemming from the lower lobe, and the lung which derives directly from the trachea. It is preferable to call the lung stemming from the lower lobe an accessory lobe. Gruenfeld and Gray,[6] reviewing the literature, cited Herxheimer[7] and Müller,[12] each reporting a case of true accessory lung in which the main bronchus came directly off the trachea. The first case was reported by Herxheimer in 1901, and Müller reported his case in 1918. In 1940, Hulse[8] reported a case of a true tracheal, accessory lung which was diagnosed by bronchogram. Our case fits into the Freedlander-Gebauer[5] classification as a tracheal accessory lung. It differs from the case reported by Hulse, as it was not only small masses of lung tissue but a whole lung. It was not situated in the mediastinum, but in the right thorax. Careful review of the English literature shows our case to be the first of a true accessory lung removed by surgery. Von Haberer[15] removed a cyst from the right interlobar fissure. Postoperative roentgenographic studies showed a bronchus leading to the region where the cyst was removed. He assumed that he had removed an accessory apical lung. In reviewing the embryology of the tracheobronchial tree, one finds many reasons for lung abnormalities. As early as in the fourth week of embryonic life, the posterior end of the outpouching arising from the caudal part of the respiratory bud assumes a bifid form. The lung buds are asymmetrical almost from the very beginning, the right being larger than the left and more caudal. The bronchial tree is formed by repeated division of the lung bud in its branches. This multiple division of the bronchial tree from the original bud gives rise to many abnormalities. Lung anomalies occur when the tracheal buds push into the mesenchyma and become partially isolated. Santy, Bérard, and Galy[13] feel that all cysts having origin in the region of the pulmonary pedicle arise from an accessory bronchial bud, or from a supranumerary lobe. In our case a complete lung with three lobes arose from a main bronchus which came directly off the trachea.

### CASE HISTORY

L. M. B., a white female infant, two months old, was seen because of a persistent cough. Chest roentgenogram revealed a homogeneous density occupying the upper two-thirds of the right hemithorax and suggesting atelectasis of the right upper lobe. Also anomalies of

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the lower cervical and upper thoracic vertebrae were noted with incomplete fusion and deformity. The first symptoms appeared two days after birth. There was a history of multiple congenital anomalies on the father's side. The mother had no communicable or other diseases while pregnant, except for an upper respiratory infection with fever during the fourth month. The patient also presented a hip deformity, with the left leg being shorter than the right.

A bronchogram was advised and performed. The bronchogram showed a stem bronchus coming directly off the bronchial tree to the right and saccular bronchiectasis in the area of the density occupying the upper two-thirds of the right hemithorax. At this time a

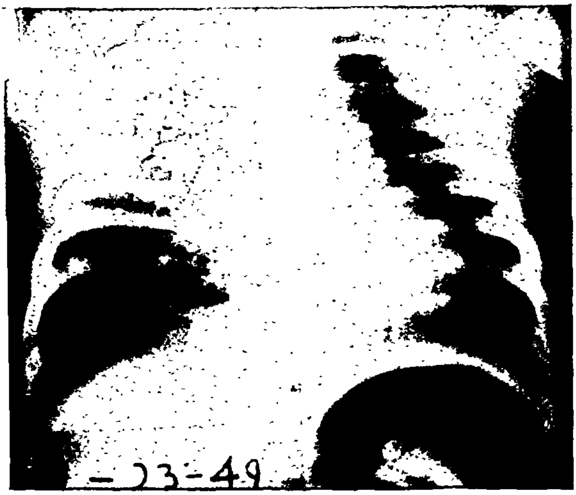


Fig. 1.—Bronchogram on Jan. 23, 1949, shows a bronchus coming directly off the trachea and saccular bronchiectasis of the area occupied by the density. Another bronchus is seen coming off the trachea below the upper bronchus.

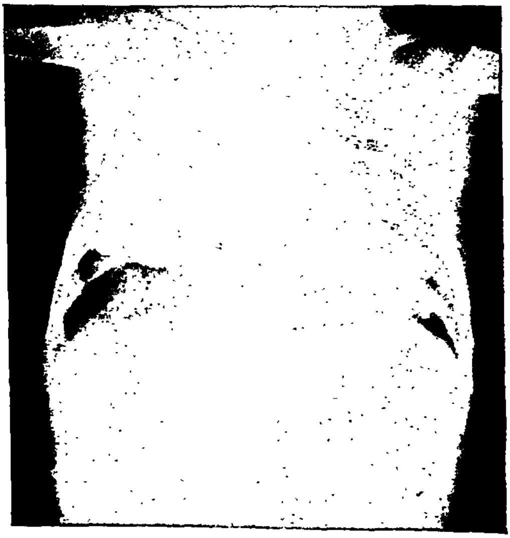


Fig. 2.—Chest film taken on May 10, 1950, shows opacification of upper three-fourths of right thorax.

diagnosis of a saccular bronchiectatic right upper lobe was made, and surgery was advised. Due to the patient's condition, conservative treatment was advised until she was older and in better condition for operation.

On May 9, 1950, the patient was readmitted to the hospital because her symptoms were worse, and fever had appeared. She could not take her feedings. Coccidioidin skin test and Mantoux test were negative. The urine showed traces of albumin. The patient received aureomycin, improved, and was discharged on June 17, 1950. She was readmitted on July 27, 1950. Her condition became worse, and she was choking easily. Surgery was advised, and a right exploratory thoracotomy was performed on July 28, 1950.

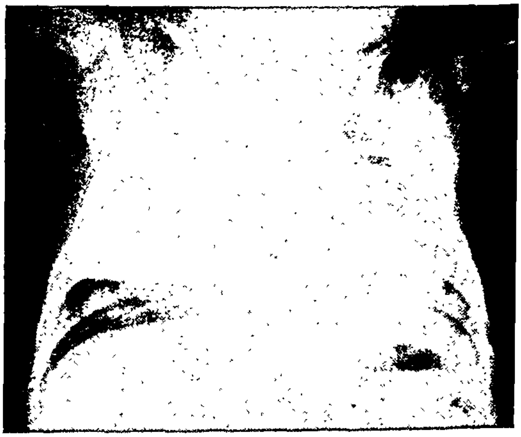


Fig. 3.—Roentgenogram taken on July 13, 1950, just prior to operation reveals essentially no change.

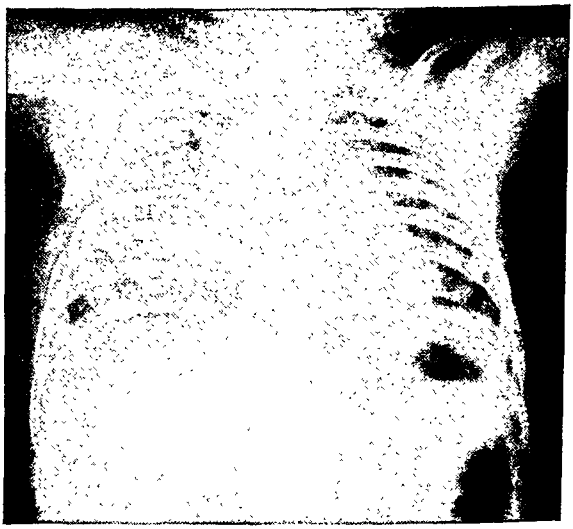
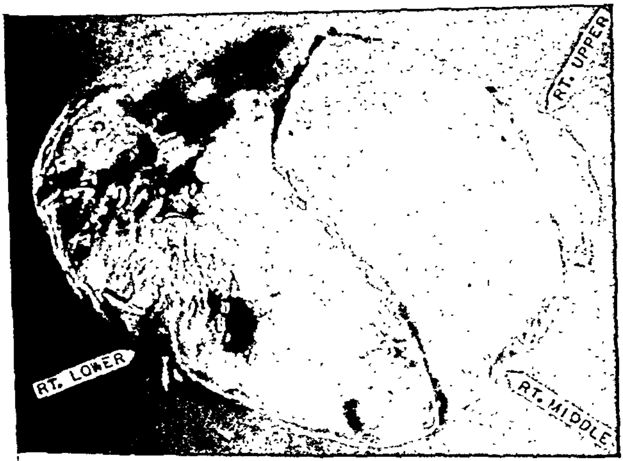


Fig. 4.—Postoperative roentgenogram revealing complete filling of right thorax by the remaining lung.

Upon opening the right pleural cavity, a large and hard tumor mass, apparently consisting of lung parenchyma, was seen and felt occupying three-fourths of the entire hemithorax. This was thought to be a cystic lobe; however, careful inspection revealed this lung to be in its own pleural cavity completely separated from the other right-sided lung which was situated inferiorly and was partially collapsed. This lower lung had a normal appearance, but it had only two lobes. It also had its completely separate, pleural cavity. The upper, accessory, cystic lung came directly off the trachea by a stem bronchus that measured approximately 3 mm. in diameter and 1 cm. in length. Two small veins each measuring 1 mm. were seen leaving the lung and following the bronchus. The arterial supply was furnished by a branch coming directly off the subclavian artery and entering the cystic lung at its apex. This artery measured approximately 2 mm. in diameter. There was no other blood supply to this lung. A right upper complete pneumonectomy was performed by the individual ligation technique. The upper parietal pleural sac was completely excised, and

Fig. 5.



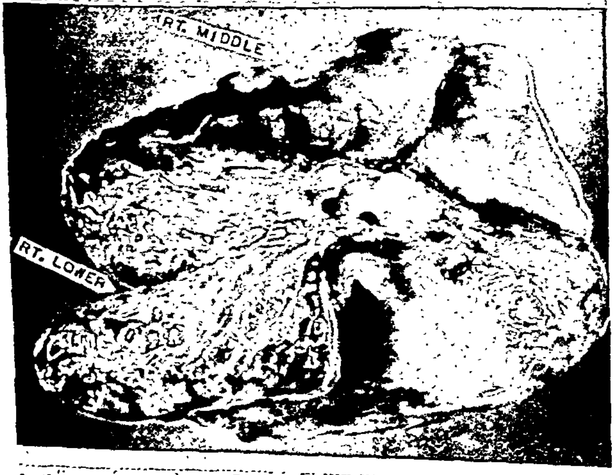


Fig. 6.

Fig. 5.—Tracheal accessory lung removed with three separate lobes, two complete fissure lines.

Fig. 6.—Cut section of one of the lobes shows the nature of the cystic parenchyma.

when the anesthetist applied positive pressure, the lower right lung completely expanded and filled the entire right hemithorax. A catheter was left in place for suction, and the chest was closed in a routine manner.

The patient had an uneventful convalescence, her respiratory complaints disappeared, and she gained weight steadily. Due to some genitourinary symptoms a retrograde pyelogram was done on Sept. 18, 1950 which revealed bilateral hydronephrosis with right hydroureter and a contracture of the vesical neck. The patient was discharged in apparently good health on Oct. 18, 1950, and was followed in the outpatient clinic. She was readmitted on Dec. 17, 1950, and a cystotomy with transverse resection of the vesical neck was performed on Jan. 10, 1951. The patient was discharged on Jan. 19, 1951, and at the present time is symptomatically and clinically well. A chest roentgenogram on Dec. 26, 1950, showed no active pathology, with both hemithoraces and lungs being normal. The patient was last seen on July 20, 1951, while admitted to the hospital for a genitourinary check-up. She is now free from respiratory symptoms.

The pathologic specimen consisted of a lung with three lobes, covered by an intact pleura. The lung weighed 82 Gm. and measured 9 by 7 by 3 centimeters. Two complete fissure lines were present. The specimen had a hilum with three major bronchi and three blood vessels. The parenchyma was filled with cystic spaces, the bronchioles were dilated, and a considerable amount of honeycombing was present.

## SUMMARY AND CONCLUSION

A case of a true accessory lung is reported.

A brief review of the embryology of the tracheobronchial tree is given.

Review of the English literature shows this to be the first case treated with success by surgical excision.

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# CARCINOMA OF INTRATHORACIC TRACHEA: EXCISION AND REPAIR BY TANTALUM GAUZE-FASCIA LATA GRAFT

## REPORT OF A CASE

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THE repair of defects of the intrathoracic trachea has recently been the subject of intensive clinical and laboratory study. Experimentally, defects have been created to simulate conditions in the human being, in whom sections of the trachea are lost after trauma, or excised as part of the treatment of strictures and tumors arising in, or secondarily invading the trachea.

From the few successes and the many failures of these various studies, two major problems have become apparent: the maintenance of anesthesia and oxygenation during the repair, and the reconstruction of the segment to be excised so that the trachea may resume physiologic function.

### ANESTHESIA

One of the most trying problems has been to devise a method of maintaining an adequate airway distal to the area being repaired, for, as the defect is made, there is a loss of the effective intraluminal pressure, as well as an escape of oxygen and anesthesia. To be sure, this situation obtains, to some extent, during the sectioning of a bronchus at pneumonectomy. But bronchial closure in that procedure is rapidly made as the lumen is opened, whereas a large defect in the trachea requires a much longer time for reconstruction.

This difficulty has been circumvented in various ways. The most obvious method is to make use of the already indwelling endotracheal tube which is in place proximal to the area to be excised. As the defect is created, the balloon on the tube is deflated and, by the combined efforts of the anesthesiologist advancing the tube and the surgeon pulling it down through the defect, the tube can be passed across the aperture and manipulated into the desired bronchus. The balloon can them be reinflated. The weakness of this method became apparent in the case to be described, when the distal tip of the tube entered one bronchus alone, thereby making difficult the inflation of the opposite lung when this was needed (usually on the operative side). In attempts to overcome this, if the tip of the tube is suspended just above the carina so as to feed both bronchi, the average balloon will not hold on a short tracheal wall cuff and may fall into the excised area, and the system would no longer be airtight. This difficulty was overcome by Belsey[1] when he added an elongated balloon to the tube to fill the excisional defect while stainless steel wire was wrapped about both balloon and trachea—which is used in the manner of a darning egg, and then covered by fascia lata.

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Carter and Strieder[2] have described what may prove to be the most practical idea. At the time of thoracotomy, rubber tubes of varying diameters and lengths are available and are inserted into the lumen through the defect until a snug fit is achieved. The tube, in place, overlaps the proximal and distal wound edges and serves as a temporary bridge while the repair is carried on about it. At the conclusion of the procedure it is removed by the bronchoscope. Perhaps if plastic or stainless steel tubes were used in this manner, they might be left in place for one to two days and serve the additional function of splinting the repaired area providing a readily available airway and exit for secretions, until sealing of the peritracheal tissues had begun. This additional tube could then be removed endoscopically.

The insertion of an oxygen-containing catheter or another endotracheal tube through the chest wall wound into the defect is of questionable value; it encumbers the surgeon, and the tidal exchange of gases in a lengthy tube extension is inadequate and may produce anoxemia.

## PRINCIPLES OF REPAIR

Certain fundamental principles of repair peculiar to the trachea have become apparent, and if any technique or material can be judged to be competent, it must fulfill most of the requirements of these principles.

The means of repair employed should vary with the condition that requires it; no single method covers every type of situation. Thus, small defects, particularly of the membranous portion of the trachea, can be bridged well by fascia lata or pleura sutured tautly over them.[7] This measure is possible as long as there is skeletal supoprt of the surrounding tracheal wall sufficient to maintain a good luminal diameter. However, large defects and excision of the entire circumference of the tracheal wall would not be corrected in this manner because of the "flutter"—or instability—of the graft when special stress is placed on the tracheobronchial tree, as in deep breathing or coughing. Moreover, it does not serve as a bulwark against the constricting effect of subsequent fibrosis or the encroachment of neighboring structures.

If no more than two or three cartilaginous rings have to be excised as, for example, in the treatment of a benign stricture, sleeve resection and end-to-end anastomosis after mobilization of the trachea has been carried out.[4] But should longer lengths of trachea require excision, there is an ever increasing danger of strain on the suture line which may separate and result in disaster. Thus, for large benign tumors or carcinomas which must have a wide margin of normal tissue to insure total excision, these limited means would be inadequate, and under the pressure of securing a closure, the surgeon might be tempted to leave a dangerously narrow cuff about disease.

As the defect enlarges, the problems of its repair increase apace, and due consideration must be paid to certain principles which we have classified as being of *immediate* and *permanent* concern.

For example, it is of *immediate* importance that the material used be relatively easily managed; no undue tension should be placed on the suture line on the trachea for fear of eventual disruption; an air- and watertight seal must be effected (air prevented from leaking out and fluid from leaking in from the surrounding structures). If this is not entirely achieved at operation it should certainly supervene within a day or two; bacteria should be kept to a minimum; and secretions must be removed adequately during the postoperative period until the tracheal defect has become well sealed off and the patient competent in this regard.

It is of *permanent* concern that re-epithelization of the defect occur and an adequate airway be maintained for the rest of the patient's life. The trachea can be considered as a "drain as well as an air duct, depending upon the ciliated epithelium to empty itself." That method of repair has merit that takes into consideration and utilizes the remarkable ability of ciliated epithelium to regenerate and spread itself across large surfaces if supported from beneath.

However, although epithelization is complete, it provides no bulwark of itself to the threat of delayed luminal narrowing as a result of scarring. This must be anticipated by providing a rigid mural support, tolerated by the body, for an indefinite length of time, for the surrounding tissues about the intrathoracic trachea cannot be relied upon to maintain the luminal diameter. Cartilaginous rings show signs of regeneration many months after the procedure by which time irreversible stenosis may well have eventuated. These rings are frequently of primitive type and may never achieve a mature contour needed to maintain a good lumen.

Various materials satisfy in part these immediate and permanent requirements as set forth above. Stainless steel and plastic tubes are easily inserted, provide a good lumen while they are in place and are tolerated surprisingly well for a fair length of time.[3] However, when re-epithelization occurs it has been found to progress *behind* these tubes so that their intraluminal surfaces are physiologically inert. If, by chance any intercurrent irritation or infection requires the removal of the tube, a debilitating stenosis at the site of repair almost always supervenes. In addition, in those instances in which carcinoma has been present, the tube in place may cover an early local recurrence of disease that might otherwise be easily observed endoscopically.

At present, work with autogenous and homogenous grafts has revealed these to be of questionable value in tumor cases. When the former are used, at least a resection of lung tissue is necessary so that a portion of uninvolved bronchial wall for grafting can be obtained to repair partial defects of the trachea.[2] However, the transplant so derived may be from an infected and/or carcinomatous lung and may possibly serve as a vehicle to return these contaminants to the patient. The homogenous grafts have been used experimentally for circumferential defects.[5] Although fresh and preserved specimens are easily handled, when defects of four or more rings are replaced by this method, late fibrosis and stenosis result and often cause the death of the animal.

For over-all utility and results in repairing large defects, from the evidence derived from human experience, the use of a metallic framework lined by a fascia lata, dermal, or pleural graft seems most promising. Belsey has had experience with the use of stainless steel wire loops, about which fascia

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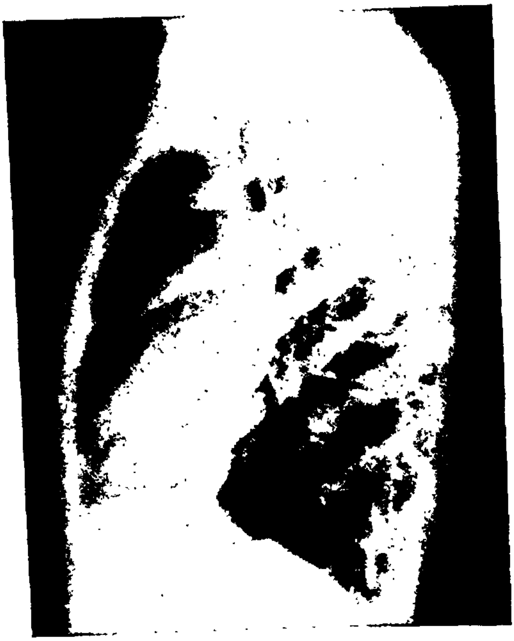


Fig. 1.—Lateral roentgenogram of chest showing oval-shaped tumor impinging on tracheal airway.

On bronchoscopic examination in July, 1950 (two and one-half years after the first attack of pneumonia as an adult), a tracheal tumor was found and a specimen for biopsy taken: this was reported as adenoid cystic carcinoma of the salivary gland type. Immediately following the biopsy, expiration and inspiration became easier, and remained so to the time of his admission to this hospital.

*Physical Examination.*—The patient was a tall, well-developed, 18-year-old white man who had acne vulgaris about the face and neck. He blushed and perspired easily and showed a marked tendency to dermatographia. The essential features of his examination were as follows: The trachea was in the midline. The chest showed equal expansion with normal resonance. The breath sounds were coarse but no râles were heard. The heart was not enlarged and the rate and rhythm were regular. The blood pressure in the left arm was 150/100, and in the right arm 170/100. A repeat recording on two occasions showed a constant level. Bilateral foot drop was present with only weak dorsiflexion of the right foot and absent dorsiflexion of the left foot. The left knee jerk was 3 plus, and the right knee jerk was 1 to 2 plus. No abnormal reflexes or changes in sensation were noted.

*Laboratory Findings.*—Complete blood count, urinalysis, and blood chemical analyses were essentially negative. The only abnormal finding was a prothrombin time of 28 lata has been wound. A primary tumor of the intrathoracic trachea was excised, and the defect managed by this technique. In another instance, a portion of trachea and carina was excised and repaired with apparently good results. Although, functionally, these repairs were adequate, 40 per cent luminal narrowing was estimated to have occurred after a period of several months. In one instance, the fascia lata infolded between the whorls of wire and required a bronchoscopic dilatation of the lumen. Perhaps the whorls of wire were placed too far apart so that the infolding was permitted.

The difficulty of infolding of the fascia lata between separated loops of wire is obviated if one uses tantalum gauze. This provides a permanent skeletal structure, well tolerated by the body, easily managed and placed with no tension on the suture line. With its reinforcement by a strip of fascia lata, a good degree of air- and watertight seal can be obtained (although some investigators using it in animals were not so fortunate). The fascia lata is eventually absorbed and the granulation tissue can insinuate itself between the spaces in the mesh and provide a bed over which the rapid epithelization from the surrounding wound edges can spread. Rob and Bateman[6] have used this method to repair a large defect in the cervical trachea following excision of a thyroid cancer which had secondarily invaded it. After excision the aperture measured 9 cm. in length and practically involved the entire circumference of the trachea except for an 0.5 cm. area at its posterior margin. They lined the tantalum mesh gauze on both sides with fascia lata, and within a few days an inpocketing or "flutter" of the inner graft necessitated bronchoscopy and the touching of its surface with the electric cautery. As this was done, the inner graft was seen to shrink against the mesh and gave no further trouble. The lumen of the trachea has been observed endoscopically and has remained undiminished for at least one year.

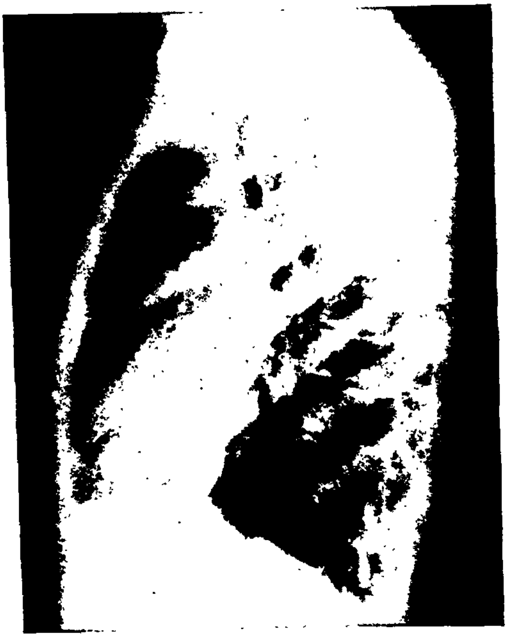
The case to be reported is that of a malignant tumor of the trachea known to be radiation-resistant, and which had produced symptoms of repeated pulmonic infections and progressive impingement on the airway. The success of the repair of the trachea after the lesion was excised can be in large part attributed to the contributions made by many of the authors mentioned above and, in particular, to Rob and Bateman for their description of their use of tantalum mesh-fascia lata graft. The diligence of the nursing and resident staff, shown in this case, was a necessary adjunct to its successful conclusion.

## REPORT OF CASE

The first admission of an 18-year-old white man to the Memorial Hospital was on Oct. 20, 1950.

*History.*—The family history was irrelevant. There was a history of polyneuritis in 1941, with residual bilateral foot drop, which was considered by a good pediatric hospital to be unrelated to anterior poliomyelitis. There has been no progression in any of his muscular symptoms since that episode. The history of his present illness dates from infancy, namely, at the age of 1½ years, when he had noisy breathing, which was thought to be caused by an "enlarged thymus." This area was treated by three or four roentgen-ray treatments, the factors of which are unknown, but no erythema of the overlying skin resulted. His symptoms disappeared following the radiation therapy. At the age of 3 years he had an attack of pneumonia which was described as severe.

He was well until the age of 15 years, when he had "pneumonia" characterized by rapid onset of fever and difficulty in exhalation, aggravated by exertion. Chest roentgenograms at this time were said to be negative. He was treated by antibiotics and was well after three weeks' bed rest. Seven months later he developed a second attack similar to the first, and required bed rest for five weeks. This attack was characterized by considerable weakness, and accentuation of difficult exhalation was again noted. Penicillin seemed to provide relief, and roentgenograms of his chest were again reported as negative. One year later, hemoptysis occurred for the first time, concurrent with a third bout of pneumonia. This subsided but returned again in five months, associated with all of the above-described symptoms.



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*Laboratory Findings.*—Complete blood count, urinalysis, and blood chemical analyses were essentially negative. The only abnormal finding was a prothrombin time of 28 seconds, the normal reading of that day being 15 to 20 seconds. The cerebrospinal fluid was found to contain 41.6 mg. of protein per 100 c.c., but no cells.

An electrocardiogram showed right axis deviation which was thought to signify hypertrophy of the right ventricle and/or vertical position of the heart.

Posteroanterior roentgenogram of the chest showed the lungs and osseous structures to be normal. There was no evidence of mediastinal widening, and the cardiac silhouette was within normal limits. However, on the lateral projection, in the midtracheal region there was a semicircular area of homogeneous density, the broad base of which was on the posterior wall of the trachea and the convex area projecting into the lumen (Fig. 1). The inferior margin of the tumor lay approximately 32 mm. above the carina, and a tracheogram showed that the tumor extended through the width of the trachea about 4 cm. in the supero-inferior diameter (Fig. 2). Lateral view of the tracheogram showed that the tumor protruded into the lumen approximately 2 cm., leaving a free airway about 5 mm. in width at its narrowest point. An esophagram was essentially negative.

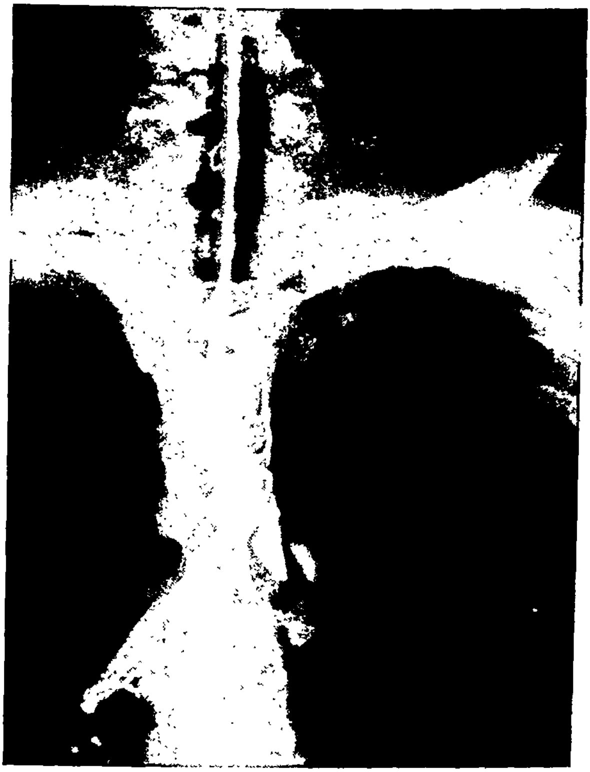


Fig. 2.—Tracheogram showing filling defect in trachea. Note its position above the bifurcation.

Bronchoscopy on September 18 showed the superior border of a yellow, nonulcerating tumor, situated 24.5 cm. below the upper dental arch. The tumor projected into the tracheal lumen for at least 1 cm. and occupied the entire posterior wall, ascending for a short distance on the right lateral wall. It was estimated at this time that about one-half of the lumen was occluded, and the bronchoscope could not be passed beyond the tumor. Vital capacity and circulation time studies were normal.

*Preoperative Preparation.*—For six days preoperatively the patient was given penicillin aerosol (100,000 units of penicillin in 1.5 c.c. of zephiran aqueous solution), four times a day. Beginning five days before operation, 300,000 units of crysticillin, twice daily; vitamin B complex; 500 mg. of vitamin C, daily; and 4.8 mg. of vitamin K, twice daily, were administered. On the day before operation, 1 Gm. of dihydrostreptomycin was started, and just prior to operation one more gram was given. Preoperative medication consisted of morphine sulfate, 1/6 grain, and scopolamine, 1/150 grain.

On October 26 anesthesia was induced with sodium pentothal and the cuff on the endotracheal tube was inflated with 4 c.c. of air. It was noticed that the airway was patent and the exchange good. Cyclopropane with 100 per cent oxygen was used throughout the procedure.

*Procedure.*—The patient was placed in the right posterolateral position and the chest and right thigh prepared with tincture of zephiran. The right pleural cavity was entered through the bed of the fifth rib after a small segment of the posterior portion of the fourth rib had also been excised. The tumor was found to lie just above the carina, on the posterior and right lateral surfaces, extending superiorly for about 3 to 3.5 centimeters. It was fairly well defined, encapsulated, somewhat irregular and nodular, and indented the esophagus without being intimately adherent to it. No gross evidence of metastases was present. The tracheal tumor was palpable through the mediastinal pleura, and a pleural flap, considered to be of adequate dimensions to cover the area to be excised, was raised. The free edge of the flap was begun in the paravertebral gutter beyond the necks of the ribs; it measured about 12 cm. in width, with its base on the mediastinal pleura overlying the superior vena cava (Fig. 3). The right vagus nerve was freed from the tracheal wall and retracted, and the azygos vein next isolated, ligated and divided. The trachea was mobilized throughout its circumference (Fig. 4, *A*) from the superior portion of the mediastinum down to the carina (Fig. 4, *B*).

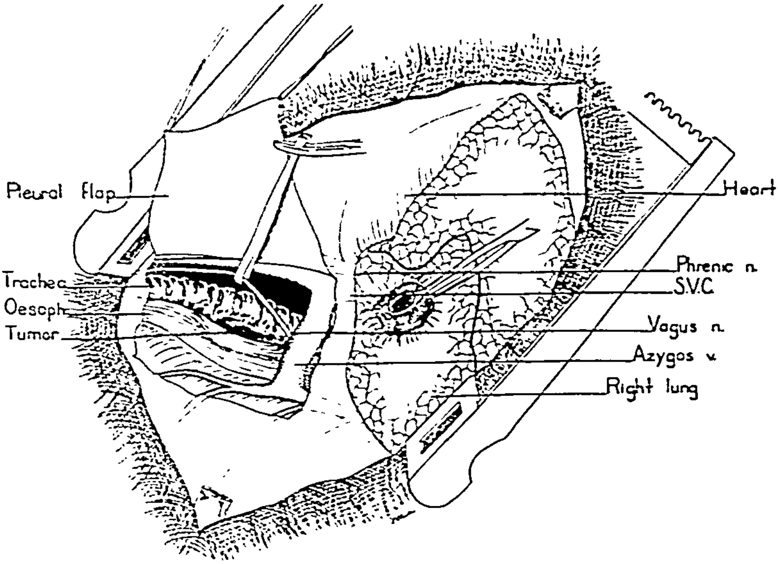


Fig. 3.—Over-all view of right thoracic cavity before azygos vein was divided.

At this time an estimation was made of the dimensions of the tumor and the expected defect to be created. The tensor fascia lata was incised vertically and a strip of fascia, about 5 by 10 cm. in diameter, was removed; this was sutured to a piece of tantalum gauze slightly smaller than its over-all surface (Fig. 5). The tantalum gauze was infolded about its periphery so that any sutures should not become weakened by possible fraying at its edges. A No. 000 plain catgut was passed through the fascia lata, then looped around the metal gauze, returned through the fascia and tied to the original suture. Multiple sutures of this type were placed about the edges of the mesh and at several points over its surface, so that there would be no shifting of the graft. For traction, a large Penrose drain was placed about the trachea, inferior to the tumor and just above the bifurcation of the bronchi.

An incision was made beginning in the cartilaginous rings, about 0.5 cm. beyond the palpable extent of tumor, and the lumen entered (Fig. 6). With the aid of a small hook retractor on the edge of the specimen (Fig. 7), the incision was developed, permitting a margin about the tumor of normal-appearing mucous membrane at least 3 to 4 mm. at its narrowest portion. As the posterior and left lateral portion of the tracheal wall was reached, the tumor-containing plaque was retracted posteriorly and the lumen widely opened.

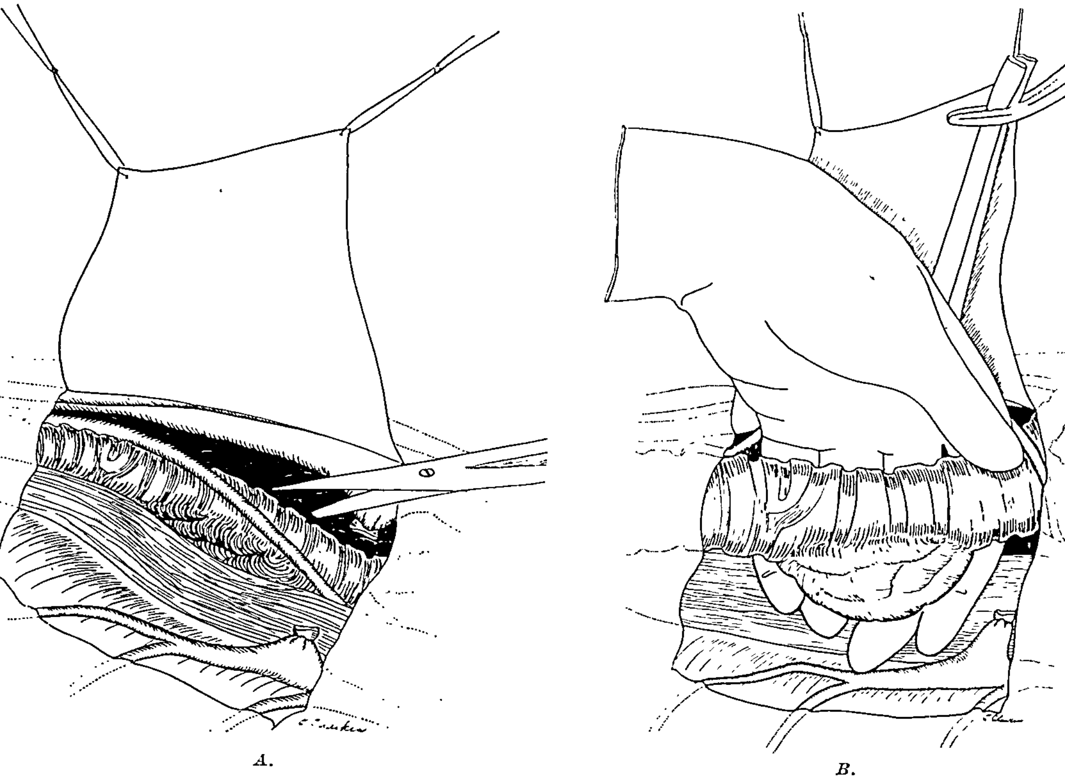


Fig. 4.—*A*, Beginning of tracheal mobilization by incision into areolar tissue between superior vena cava and trachea. *B*, Trachea and tumor isolated. Note division of azygos vein and retraction of vagus nerve.

The anesthesiologist, who had been maintaining respirations manually because the patient had stopped spontaneous breathing for some time, deflated the cuff, and a Kelly clamp was inserted into the orifice to pull down the lower portion of the endotracheal tube, pass it across the defect and into the left main bronchus (Fig. 8, *A*). This procedure was relatively easy, but when the anesthesiologist attempted to reinflate the cuff it was discovered that the clamp had made a rent in it. It thus became apparent that a closed system would have to be abandoned, and consequently the tumor was excised expeditiously. The resulting defect measured about 7 by 3 cm., and approximately one-half of the anterior portion of the tracheal cartilages was left intact.

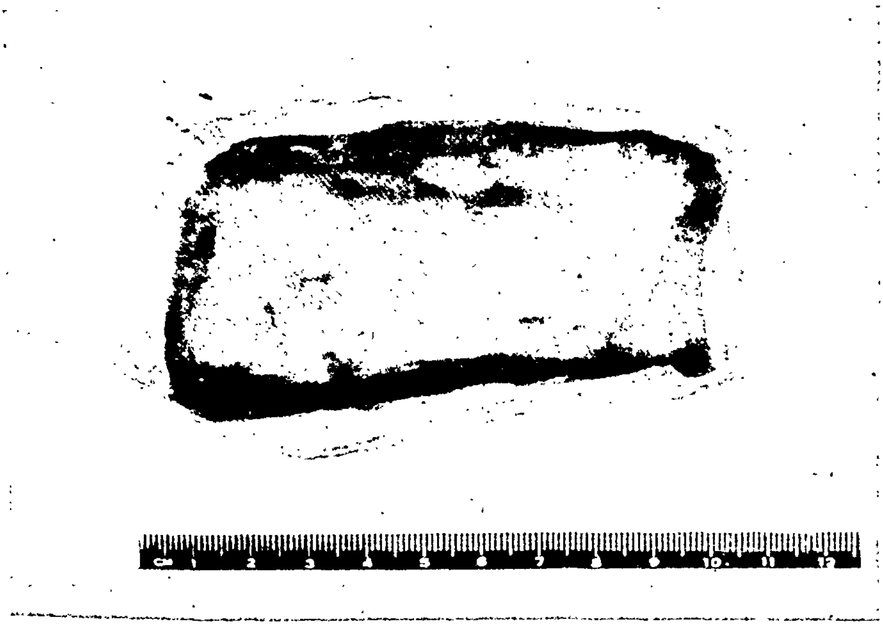


Fig. 5.—Tantalum mesh gauze and fascia lata sewn to it over multiple points. Note rolling of edge of mesh to prevent fraying and slipping of sutures at its margin.

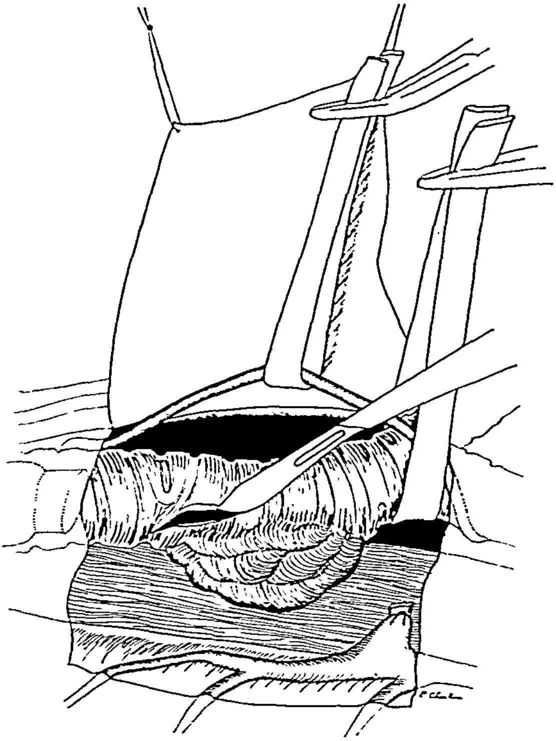


Fig. 6.—Incision into trachea. Penrose drain just above tracheal bifurcation. Right vagus nerve retracted.

At this point the patient became cyanotic, probably as a result of oxygen loss through the defect, coupled with diminished pulmonary excursions from the poor control of the closed system. Two maneuvers were used to overcome the problem of anoxemia: The endotracheal tube was drawn upward until its tip rested above the carina, and by tightening the Penrose drain that was in place above the carina about the trachea, occluding its walls sufficiently about the tube, a substitute for the broken balloon cuff was achieved (Fig. 8, *B*). The right lung, which was collapsed during the procedure, was inflated and employed for oxygen. In this manner the procedure progressed somewhat haltingly, for each time the right lung was collapsed to permit suturing, cyanosis would soon begin, so that sutures could be placed only in groups of four or five.

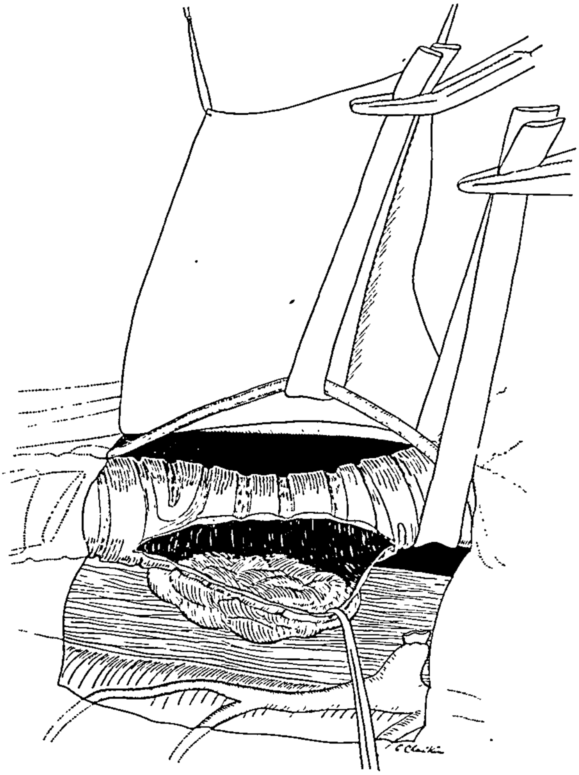


Fig. 7.—Retraction of tumor specimen to permit view of its interior extent.

The stent, composed of the fascia lata graft and the tantalum mesh, was held outside the thoracic wound in the position it would assume about the trachea, and a row of No. 000 plain catgut sutures was begun, uniting fascia lata and the superficial portions of the tracheal wall for a distance of 0.5 cm., beyond the edge of the left side of the defect in the trachea (Fig. 9). This was made feasible by rotation of the trachea throughout its long axis so that its opposite surface presented more readily. In addition, several No. 000 chromic catgut sutures were passed through the tantalum mesh and through corresponding portions on the tracheal wall about 3 mm. from its cut edge (Fig. 10). The graft was slid into position along these "guy ropes," and the sutures tied individually. The tracheal torsion was relaxed and the mesh and fascia lata were curved to cover the defect. The fascia lata was tacked to the tracheal wall by means of multiple silk sutures, in the form of a patch. As soon as this was completed, the trachea was relatively airtight, and the endotracheal tube could be used to better advantage for oxygen and anesthesia without

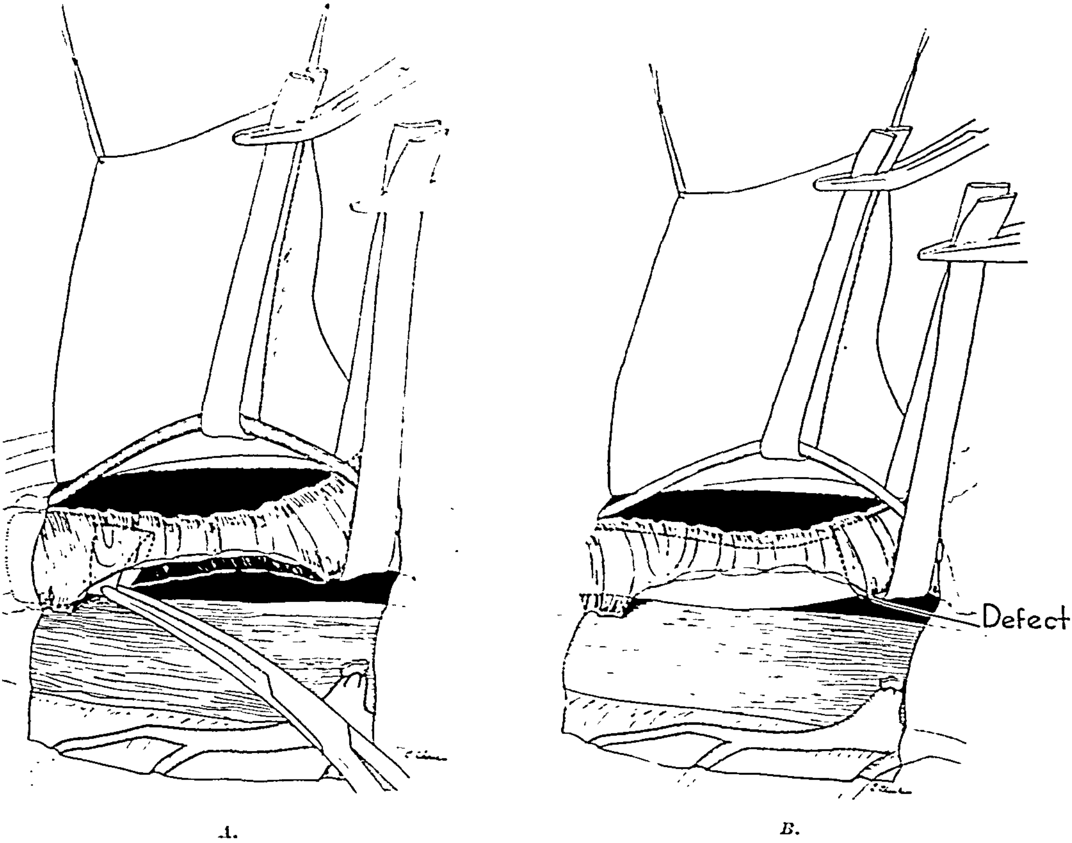


Fig. 8.—*A*, Clamp shown pulling distal tip of intratracheal tube with cuff deflated, down into the left main bronchus. *B*, Tube in place. Its position was maintained by the Penrose drain just above bifurcation because the balloon kept slipping out into the defect.

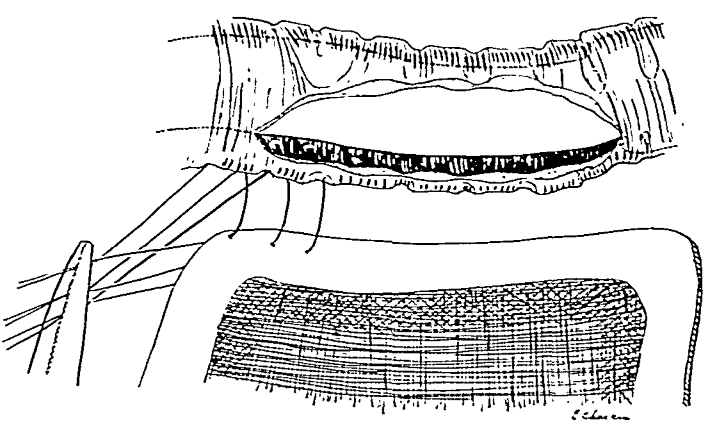


Fig. 9.—Beginning of posterior row of sutures through edge of fascia lata portion of stent.

the aid of the Penrose drain's circumtracheal compression. Retraction on the right vagus nerve was relaxed and the pleural flap that had been previously prepared was sutured over the graft with interrupted silk sutures, but not tacked too intimately to the inferior margin of the mediastinal defect so as to allow for possible egress of air (Fig. 11). A

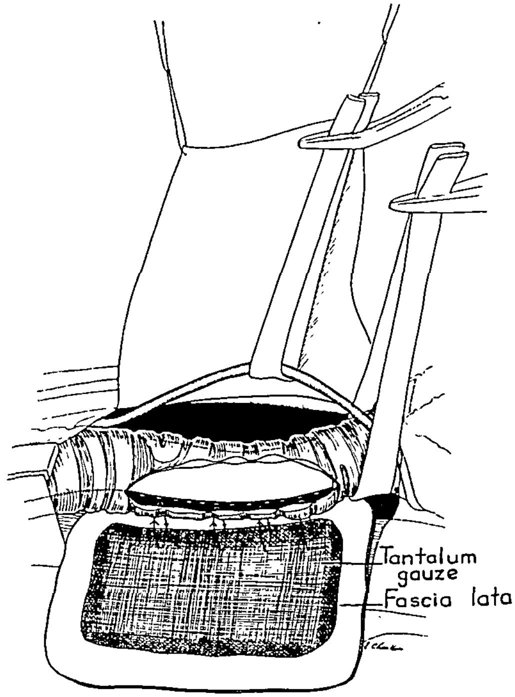


Fig. 10.—Interrupted sutures bringing tantalum mesh to edge of defect after posterior row of fascia lata sutures had been placed.

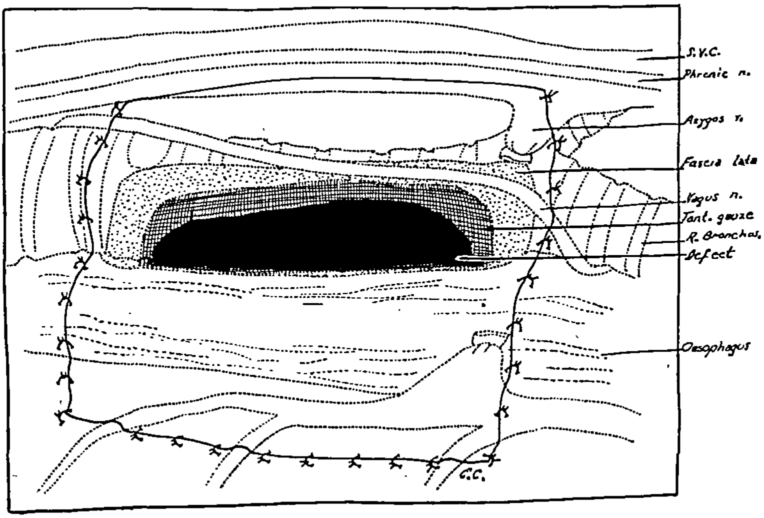


Fig. 11.—Completed repair demonstrating the pleural flap (in solid line); the fascia lata (stippled); the mesh (cross-hatched); and the defect (solid black).

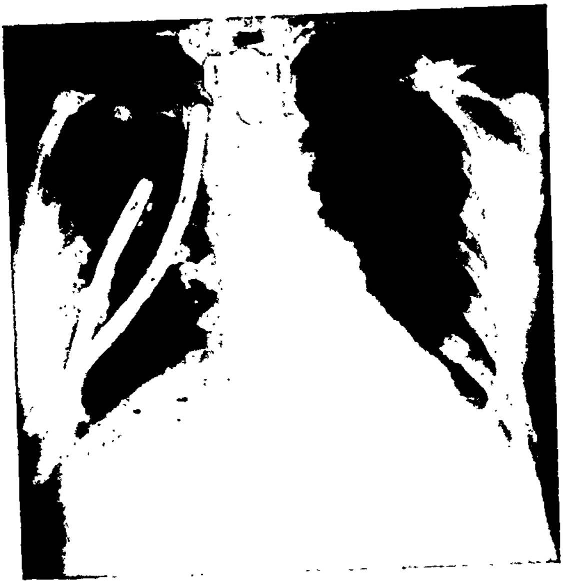
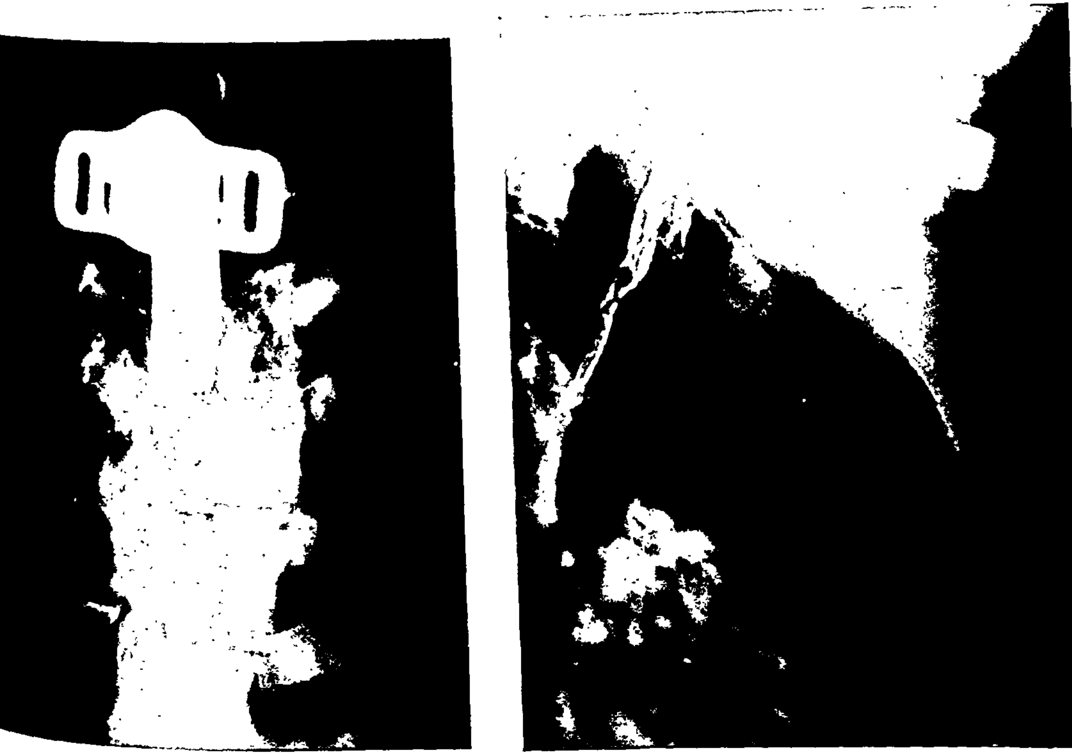


Fig. 12.—Roentgenogram of chest showing upper and lower underwater drainage tubes and tracheotomy tube in place (first postoperative day).



A.

B.

Fig. 13.—*A*, Front view of trachea showing tracheotomy tube and tantalum mesh. *B*, Lateral view of trachea showing tracheotomy tube and mesh. Note lack of diminution in size of tracheal lumen, and that upper margin of tracheal repair could be visualized through tracheostome when the tube was removed.

portion of the esophageal wall at the superior margin of the graft was sutured to the trachea to seal its upper end. Two hundred thousand units of penicillin, 1 Gm. of streptomycin, and 10 c.c. of saline were injected into the pleural cavity, and eucupine in oil was injected into the fourth, fifth, and sixth intercostal nerves. Two thoracotomy drainage tubes were inserted: one through the third intercostal space anteriorly and another below the thoracotomy wound in the eighth intercostal space. These were connected with individual underwater drainage bottles and the chest wall closed in layers.

The patient was then placed in the supine position and a tracheotomy performed with the insertion of a No. 8 tracheotomy tube. The purpose of this tracheotomy was to insure an avenue of suction, but in addition it was to decompress the tracheobronchial tree during exertion and coughing, resulting in less intraluminal air pressure referred to the suture line. His color was good and he was returned to the ward almost awake.

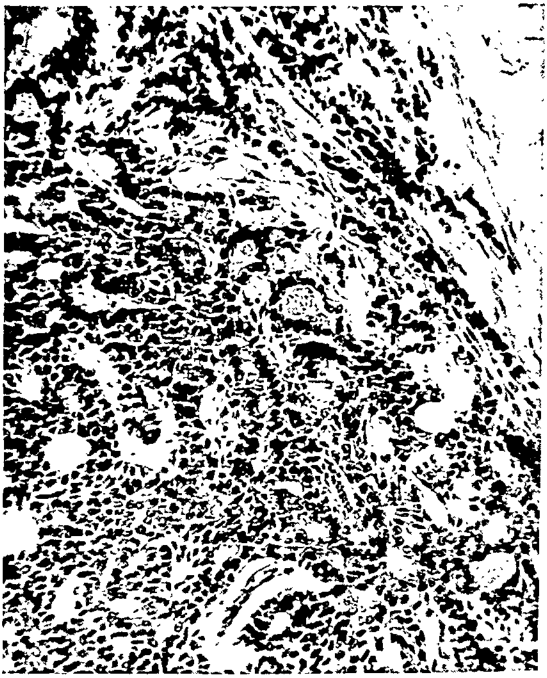


Fig. 14.—Photomicrograph of tumor called adenoid cystic adenocarcinoma, salivary gland type.

During the procedure he received 500 c.c. of normal saline and 1,000 c.c. of blood. His blood pressure at the onset of anesthesia was 130/70 (as compared with 165/105 obtained in his conscious state), and remained fairly static throughout.

*Postoperative Course.*—The patient was placed in an oxygen tent, and given 100,000 units of penicillin every three hours; 0.5 Gm. of dihydrostreptomycin every six hours; vitamins K, B, and C in preoperative quantities; and penicillin spray by De Vilbiss syringe through the tracheotomy tube. In addition, tracheal aspirations were done as needed. He ran a surprisingly uncomplicated postoperative course. Bubbling never occurred through the underwater drainage tube at any time, and both fluctuated for several days (Fig. 12). The suction material obtained from the tracheotomy was white and purulent for at least three postoperative days, and on the fourth day it began to clear. His temperature ranged from about 101° to 103° F. for the first postoperative week, gradually returning to normal on the sixteenth postoperative day.

Portable roentgenograms of the chest postoperatively showed the left lung to be entirely clear, and the right lung to have several densities (thought to be old lipiodol deposits) scattered throughout the right central lung field. In addition, there appeared to be a small collection of fluid in the right pleural cavity, and on the fifth postoperative day this was tapped, yielding only 20 to 30 c.c. of clear fluid, and 300,000 units of penicillin were injected into the pleural cavity. Streptomycin was discontinued on his fourth postoperative day from which time 150 mg. of aureomycin, every six hours, was given. On the ninth postoperative day, the lower underwater drainage tube was removed; on the tenth day he was moving about the room, and on the twelfth day the second underwater drainage tube was removed.

On the twentieth postoperative day, the tracheotomy tube was removed to be cleaned, and it was possible to see the upper one-third of the defect in the tracheal wall (Fig. 13, *A* and *B*). At that time a sharp edge of the defect could be seen with no signs of inflammatory reaction about it. A yellow background was observed through the mesh, undoubtedly representing the fascia behind it. This excellent chance to observe healing of the graft in situ was utilized from that day until the twenty-third postoperative day when definite signs of "pinking" were observed through the mesh as the granulation tissue appeared. In addition, it was noted that epithelial ingrowths along the edge of the defect were beginning to decussate the mesh. On the thirtieth postoperative day, granulation tissue budding through the mesh was clearly evident, and the tracheotomy tube was removed permanently.

The patient was discharged from the hospital on the thirty-first postoperative day, and since that time has been relatively asymptomatic. Bronchoscopy, three months postoperatively, revealed no trace of any tantalum mesh and epithelization appeared to be about 100 per cent complete. Repeat bronchoscopic examinations were made every two months and showed that the lumen had maintained its diameter completely and that there was no evidence of tumor. His latest bronchoscopic examination on Oct. 31, 1951 (one year postoperatively), revealed no recurrence of the tumor or luminal narrowing. Four months later there was still no evidence of disease.

*Pathologic Report.*—The pathologic specimen measured over-all 3 by 3 by 2 cm. and showed an adenoid cystic adenocarcinoma, salivary gland type (Fig. 14). Dr. Fred Stewart states, "Of course, the behaviour of such a lesion in the trachea is *not* well documented, but it is extremely sluggish, and the extent to which this tumor can metastasize is virtually unknown."

## SUMMARY

A case of carcinoma of the trachea, with excision and repair of the defect with tantalum mesh gauze and fascia lata, is reported. The patient is alive and without evidence of disease or change in tracheal lumen size sixteen months after operation.

A brief reference is made to various methods described in the literature which have been used to overcome the physiologic and reparative difficulties attendant upon such procedures, and their application to this case noted.

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# LIPOTHYMOMA: AN UNUSUAL BENIGN TUMOR OF THE THYMUS GLAND

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BENIGN tumors of the thymus gland are extremely uncommon. Adenomas, fibromas, lipomas, and myxomas are said to comprise the majority of this group. Benign encapsulated mixed tumors composed of inextricably intermixed thymic and fatty elements are virtually unknown. As far as we are aware, an unequivocal example of such a neoplasm has been reported only once.[2] As in the case to be reported here, the patient was a child, and the tumor was not associated with myasthenia gravis or any other abnormality. Because of the unusual character and apparent rarity of this type of benign tumor of the thymus gland, the present case is reported in considerable detail.

## CASE REPORT

P. O., a 10-year-old white girl, was admitted to the hospital during November, 1946, because of paroxysmal coughing of ten weeks' duration. She also complained of raising more sputum than normal and on two occasions the sputum had contained blood. Roentgenograms of the chest, taken one month prior to admission, showed atelectasis of the right middle lobe. The child had never suffered from dyspnea, although she became easily fatigued while playing. There was no clinical evidence of tuberculosis or of myasthenia gravis.

On admission her temperature was 99.6° F., pulse 80, and respirations 20. She was a slightly built girl, appearing younger than her stated age. A few anterior and posterior cervical lymph nodes were palpable. Anteriorly a region of increased dullness extended from the right of the sternum to the nipple line, and from the second to the fifth interspaces.

A chest roentgenogram revealed the presence of a mass in the anterior mediastinum. It was oval in shape, sharply outlined, rather dense, and extended laterally toward the right (Figs. 1 and 2). The leukocyte count was slightly elevated, being 10,400 per cubic millimeter. Nothing else of note was found.

Right anterior thoracotomy was performed three days after admission, and a well-encapsulated solid tumor was exposed in the anterior mediastinum. It was attached to the lower lobe of the thymus, and extended from the pericardium into the right thoracic cavity, compressing and rendering atelectatic the middle lobe of the right lung. The tumor was readily and completely removed.

*Pathologic Report.*—The specimen received in the laboratory consisted of a firm, encapsulated, oval tumor mass measuring 12.0 by 9.0 by 4.5 cm. in size, and, after fixation, weighing 170 Gm. The capsular surface was thin, smooth, glistening and transparent, but a few petechial hemorrhages, together with a few adherent fibrous tags, were present. On section, the surfaces exposed by cutting were firm, rubbery and pale yellow (Fig. 5). While the color was somewhat suggestive of a lipoma, the firm fibrous consistency of the tumor indicated that the mass was something more than encapsulated adipose tissue.

The entire specimen was fixed in 10 per cent formalin solution and blocks of tissue to be prepared for microscopic study were fixed both in 10 per cent formalin solution and in Regaud's solution. The sections were stained with hematoxylin and eosin.

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†Dr. Ehler died on Nov. 5, 1951, after a brief illness.

*Microscopic Description.*—The tumor was found to be an unusual type of thymoma which was infiltrated throughout by normal-appearing adult adipose tissue and varying amounts of fibrous connective tissue. Sections taken from various portions of the growth, not only superficially but also deep within it, showed this admixture of thymus gland and fibro-adipose stroma to be uniformly present throughout the neoplasm, though the relative proportions of each varied in amount (Figs. 6 and 7). The thymic tissues were readily recognizable, being composed of accumulations of thymic lymphocytes in which were varying numbers of well-differentiated Hassall's corpuscles (Figs. 8, 9, and 10). The only variations from normal

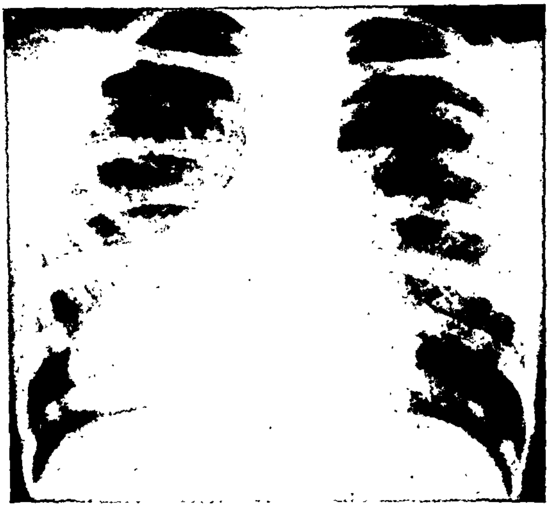

Fig. 1.

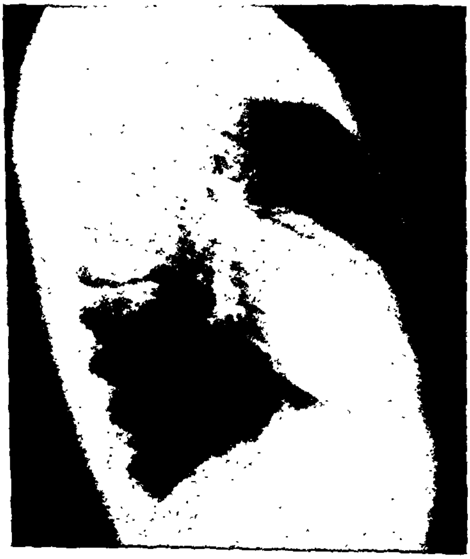

Fig. 2.

Figs. 1 and 2.—Roentgenograms taken before operation, showing the tumor as a large, well-outlined mass which appears to arise in the anterior mediastinum and to extend into the right pleural cavity.

thymic parenchyma were the distorted character of the lobules and the abundance of thymic tissue which appeared definitely increased in amounts. The lobules appeared either as isolated islands or as irregular narrow or broad anastomosing bands separated from each other by fatty stroma. There was also some loss of demarcation between cortical and medullary zones. While the stroma was composed chiefly of adipose tissue, it was mixed here and there with loose fibrous tissue. In some places the connective tissue actually predominated, being present either as bands of collagen similar to those which might be found in a fibroma,

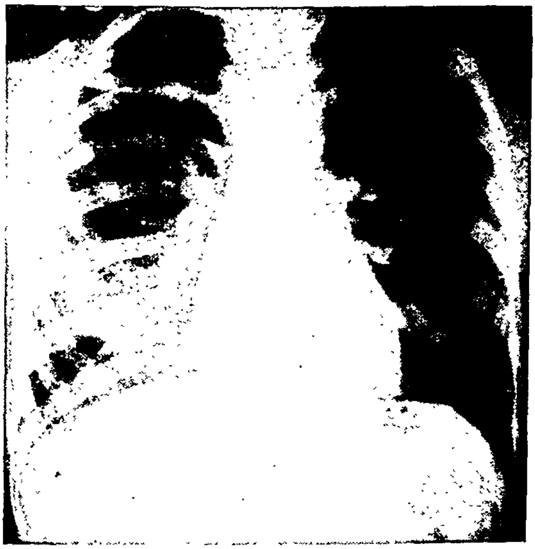

Fig. 3.—Roentgenogram taken after operation. The tumor is no longer present, and the thorax appears normal.

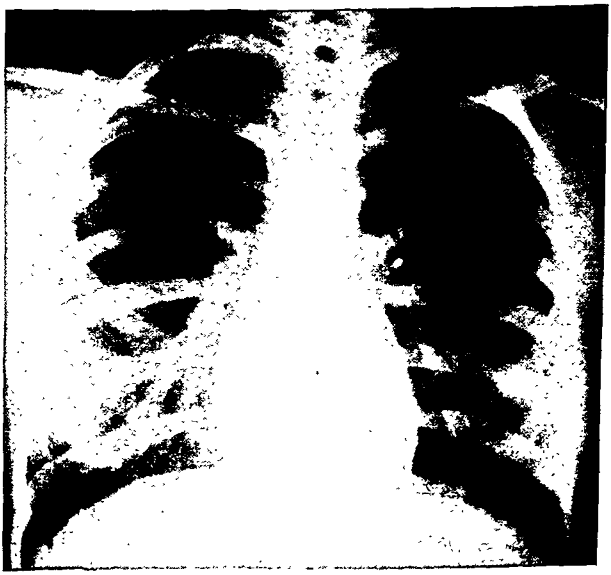

Fig. 4.—Roentgenogram taken four years after the tumor was removed. There is no change from the appearance of the thorax as shown in Fig. 3.

or as loose areolar tissue containing accumulations of fat cells within it. Sometimes the stroma was composed of adipose tissue alone. The most typical stroma was formed by both fibrous and adipose tissues with the fibrous tissue somewhat more predominant adjacent to the thymic lobules, while fat tissue was more abundant in the stroma further removed from the thymic elements. Thus, although the architectural pattern of the thymic tissue appeared altered and distorted by infiltrating fat and fibrous tissue, there was none of the disorderly tissue array that characterizes a malignant neoplasm.

The thymic tissues themselves appeared more abundant than normal suggesting that, as the neoplasm grew, thymic as well as adipose and fibrous tissues participated in the growth. The thymic tissue, however, became stretched or compressed as the neoplasm grew, with the result that it became a distorted, malformed structure within the tumor mass.

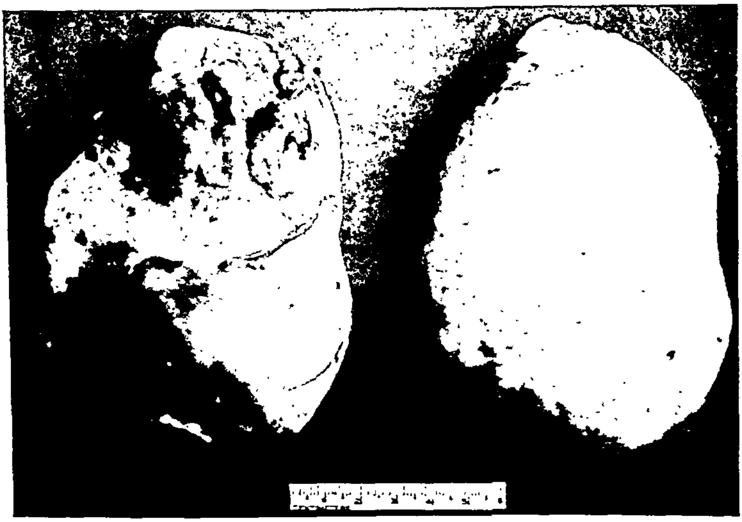

Fig. 5.—Photograph showing the capsular (left) and cut surfaces of the mediastinal tumor. The neoplasm is well encapsulated, solid, and homogeneous. Note large vessels in capsule and foci of subcapsular hemorrhage.

The over-all picture was that of a benign, well-encapsulated tumor composed of widely radiating strands of thymic tissue separated by proliferating fibro-adipose stroma. As indicated above, it appeared that both glandular and stromal elements participated in forming the benign neoplasm, thus making it a kind of mixed tumor, a fibro-lipo-thymoma or simply a lipothymoma, since adipose and thymic tissues predominated.

The child's postoperative course was entirely uneventful. Within four days the middle lobe of the right lung was completely re-expanded (Fig. 3) and she was discharged on the tenth hospital day. Periodic follow-up physical examinations during the next five years have shown no suggestion of recurrence of the tumor (Fig. 4), and there have been no signs or symptoms even suggestive of residual disability.

## DISCUSSION

Since this girl has now lived for five years without any indication of recurrence, it seems reasonable to conclude that this unusual thymic tumor was entirely benign. Its large size, the symptoms it produced, and the roentgenographic findings presented no difficulty in establishing the presence of a mediastinal mass which needed to be removed. The operation was, therefore, completely successful.

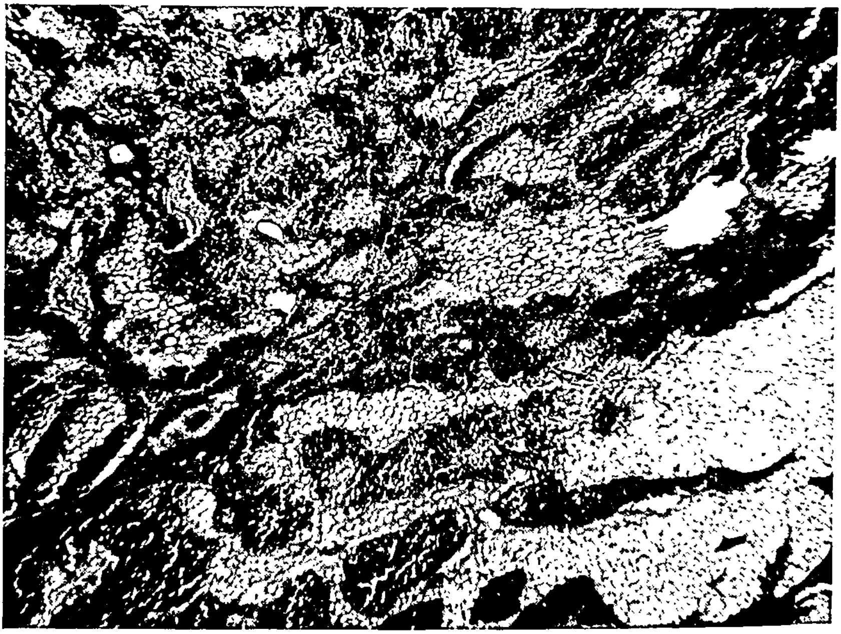

Fig. 6.

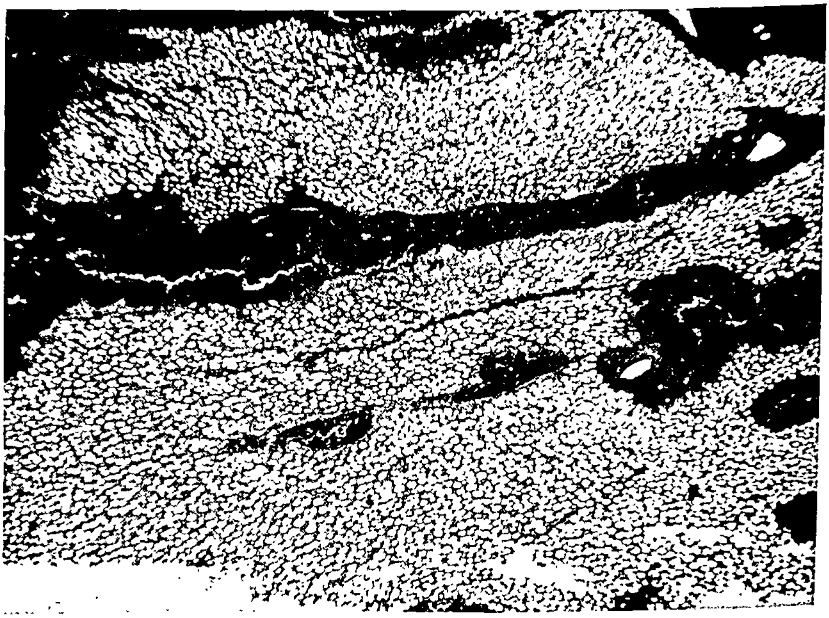

Fig. 7.

Figs. 6 and 7.—Low-power views showing the structure of the neoplasm. Note irregular, distorted anastomosing cords of thymus gland surrounded by narrow bands of collagen, and embedded in varying amounts of adipose tissue. (×30.)

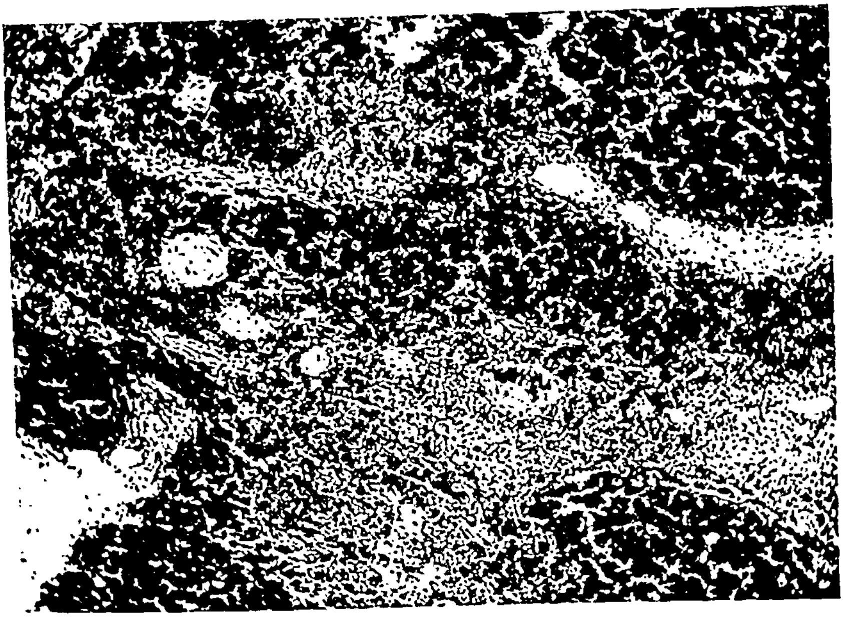

Fig. 8.

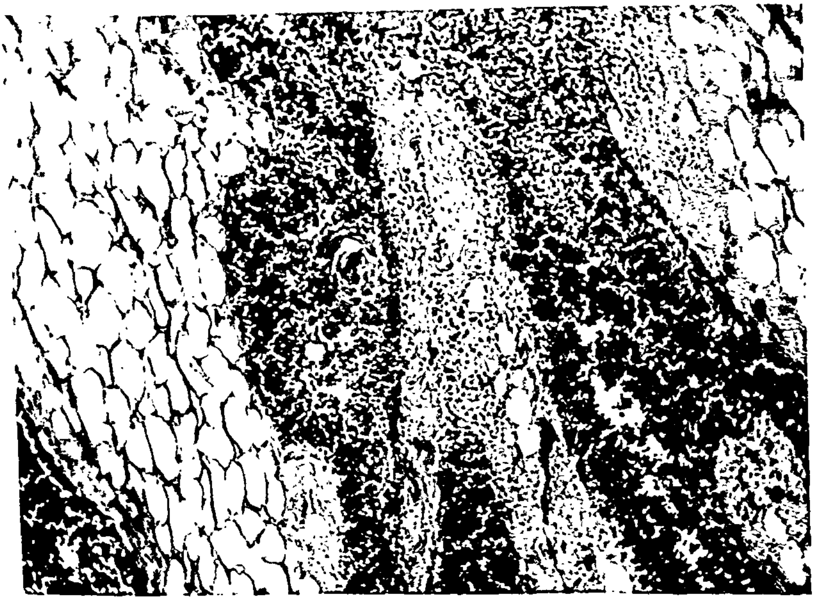

Fig. 9.

Figs. 8 and 9.—Sections showing typical thymic parenchyma embedded in adipose tissue. In Fig. 9 the presence of dense collagenous tissue adjacent to the thymus gland is clearly seen. Note typical Hassall's corpuscles. (×150, ×180.)

A review of the literature brings to light only one other unequivocal instance of an essentially similar tumor.[2] A few authors, in their descriptions of thymic tumors, suggest that they may have observed this type of thymic growth,[3, 14] but only the report of Andrus and Foot clearly describes pathologic changes that in most respects correspond to those observed by us. The size of the tumor in their case, 2,235 Gm., made it the largest thymic tumor ever recorded. In the years since their report no one else has encountered, or at least reported, any thymic tumor resembling this one.

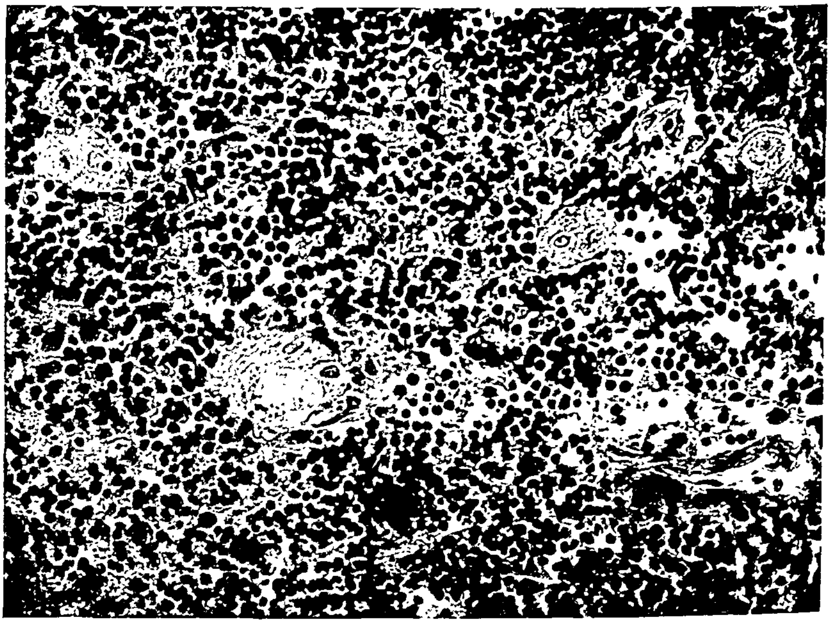

Fig. 10.—High-power view to show the typical thymic structure of the gland. Note thymic lymphocytes and reticulum and Hassall's corpuscles which were present in all parts of the tumor. (×550.)

Actually the changes of so-called involution of the thymus are characterized by atrophy of the gland with separation of the thymic lobules by a loose alveolar stroma. Descriptions of histologic sections from such glands, then, do not seem to differ significantly from the histologic features of this tumor. It has been noted also that with increasing age the adipose tissue component of the thymus increases proportionately, while the parenchyma on the other hand is decreasing.[13] Consequently, some question may be raised as to the nature of this large encapsulated growth. Is this mass really a neoplasm, or simply a form of fibro-adipose infiltration or an abnormal type of involution? The fact that the mass was circumscribed, localized, and of such large size makes it obviously a neoplasm, for it grew as an independent entity at a greater rate of speed than the normal tissues about it, ultimately forming the solid encapsulated tumor found at operation.

Is it a lipoma or a thymoma? This question cannot be so readily answered. In the strictest sense, a lipoma is an encapsulated mass of adipose tissue or fibro-adipose tissue which increases progressively in size, grows independently of the surrounding tissues, and extends laterally by pushing adjacent structures to one side without infiltrating them. When various other components are also present, such as fibrous tissue, vascular tissue, etc., such terms as fibro-lipoma or angio-lipoma may be employed. When the adipose tissue appears less prominent than the fibrous or vascular elements, then the terms lipomatous fibroma or lipomatous angioma (lipo-angioma) may be employed. This tumor was a firm, rubbery, pale yellow neoplasm, ovoid in shape, encapsulated, and more like a fibromatous tumor than the soft, lobulated, bright yellow growth that is the typical lipoma. Therefore, this neoplasm did not seem to be a lipoma, although it did not resemble a thymoma either.

A thymoma, on the other hand, has always been difficult to characterize or classify. This is especially true of the malignant group where either lymphoid or epithelial elements, or both of them together, may participate in the neoplastic growth. With regard to benign thymic growths, the greatest difficulty is to differentiate between hyperplasia and neoplasia. Benign thymomas, however, characteristically possess a distinct capsule and histologically exhibit a distorted thymic structure often with a loss of cortico-medullary boundaries. Thickened trabeculae are generally encountered. Since most benign thymic tumors are adenomas, they are generally composed of solid masses of small round cells resembling lymphocytes infiltrated with thymic reticulum and Hassall's corpuscles. The observation has been made that fat characteristically does not infiltrate these tumors.[6] Consequently a benign thymic adenoma is an encapsulated,, circumscribed tumor composed of structurally disarranged thymic elements often with dense dividing trabeculae, and usually devoid of adult fat cells.

The growth in our case, then, is characteristic of a benign thymic tumor which, however, is not a simple adenoma. For histologically the growth proved to be composed of thymic, adipose, and some fibrous tissues, thus making it a mixed tumor. The lymphoid elements, Hassall's corpuscles, thymic lobular arrangement, and thymic reticulum, unequivocally indicated its thymic nature. The noteworthy feature, as can be seen from the illustrations, is the marked and abundant admixture of thymic tissue with fibro-adipose stroma.

How shall a tumor of this type be named? Since the histologic nature and origin of many thymic tumors is still a subject of controversy, the organ designation with the suffix "oma" has served as the most satisfactory compromise for an admittedly unsatisfactory method of classification. This situation is not unique, however. For example, some tumors of the liver are still referred to as hepatomas; some which arise in the kidney are called hypernephromas; and those that arise in glial tissue are gliomas. It would seem appropriate, therefore, to designate this tumor a lipothymoma. If the expression, fibrolipoma, can mean a lipoma with fibrous tissue elements in it, then a lipothymoma may be interpreted as a thymoma with adipose tissue elements in it. The term thymolipoma might also be used, though thymolipoma emphasizes the lipomatous character of the growth rather than the associated thymic element. This unusual tumor, then, is considered to be a lipothymoma, for it is so obviously composed of glandular and stromal tissues which grow as a true tumor. Moreover, both of these tissue elements appear to have participated in the formation of the neoplasm.

While many thymomas, both benign and malignant, may be associated with myasthenia gravis, in this case, and in that of Andrus and Foot, they were not. It is also noteworthy that both patients were children, one a 10-year-old girl, the other a 13-year-old boy. Both tumors were composed chiefly of adipose or of fibro-adipose tissue growing intimately and coordinately with thymic tissue. Although other unambiguous instances of similar neoplasms have not been recorded, or at least cannot be readily found in the literature, mention should be made of the report by Norris,[14] who recorded four instances of tumors associated with myasthenia gravis. His Case 1 was that of a 37-year-old woman with this disorder. At necropsy a thymic tumor weighing 57 Gm. with two cervical projections was found. The major mass of the tumor had a solid cellular structure, but sections from one of the projecting masses, designated as the left cervical neck, revealed a histologic picture similar to that of the tumor in our case. The tumor in his second case was in a 33-year-old white man who also had myasthenia gravis. This tumor weighed 17 Gm., and the histologic pattern was described as showing a lobular arrangement of thymic tissue with tonguelike projections separated by loose areolar tissue and dense fibrous tissue. Possibly both these tumors belong in this category of lipothymoma.

The thymic nodules reported by Alter and Osnato[1] do not resemble this entity, lipothymoma, as the title of their own paper clearly indicates although Andrus and Foot considered some similarity to exist. Furthermore, the tumor reported by Danisch and Nedelmann[8] is also in no way similar to this tumor type though it seems so regarded by Andrus and Foot. A translation of the title of their article clearly shows the dissimilarity. "A malignant thymoma in a 3½-year-old child with unusual metastases to the central nervous system." Perusal of this paper shows the histological pattern of their tumor to be clearly different.

It was thought that perhaps some of the recorded lipomas of the anterior mediastinum might be, in reality, tumors of this type.[5, 10, 11, 16] Possibly the thymic nature of yellow fatty-appearing tumors overlying the pericardium beneath the lower end of the sternum was simply overlooked. Beatson[3] records the case of a 45-year-old man in the anterior superior portion of whose mediastinum was an orange-sized lipoma. It was thought possible that this tumor might contain thymic elements. Unfortunately, no confirmatory microscopic studies were made. None of the other of more than fifty examples of mediastinal lipomas contains any reference to thymic tissue in fat. To be sure, histologic reports do not accompany all of the cases, yet, where recorded, mention is made only of mature fat, sometimes with an admixture of their connective tissue elements.

Finally, reference is occasionally made to lipomas of the thymus. However, no definitive bibliographic reference with histologic characterization could be located. For example Crotti[7] in cataloging tumors of the thymus states, "A lipoma has been reported by Wadde, Münchmeyer and Müller." Yet his bibliography gives no reference which can be checked. Karsner[12] mentions that lipomas of the thymus have been reported. The only reference he cites in which lipomas are mentioned is that of Margolis.[15] Yet all this latter author says is that lipomas have been reported occasionally. His review and that of Bell[4] have recorded most of the thymic tumors in the older literature, but neither of them cites a reference to a report of a lipoma of the thymus gland. Furthermore Geschickter,[9] in an extensive review of lipomas, fails to record a lipoma of the thymus, although notations of such rare occurrences as a lipoma of the larynx and of most internal viscera is made. To be sure, an exhaustive search of the literature of the nineteenth century might turn up a concrete example. However, it is not certain that such a report would bear careful scrutiny. Was such a case from the mediastinum only? How was it determined that this lipoma was located in the thymus? Was it by gross observation alone? Was an involuted fatty thymus called a lipoma? Was an encapsulated mass of adipose tissue found in a thymus gland? It is even possible that this and other vague references to lipomas of the thymus refer to the entity herein described as lipothymoma, although we have been unable to verify this supposition.

Thus only one other unequivocal similar case is recorded in the literature[2] and two other plausible instances,[14] which were dissimilar in that they occurred in adults and were associated with myasthenia gravis. Of course tumors of the thymus are uncommon, and benign thymic tumors make up only a small percentage of this group. For that reason this entity, lipothymoma, will at most be encountered only infrequently.

## FINAL SUMMARY

A rare benign thymic tumor composed of thymic and fibro-adipose tissues is described and its nature and possible origin are discussed. The neoplasm developed in the anterior mediastinum of a 10-year-old girl and was unassociated with myasthenia gravis. It is suggested that it be designated a lipothymoma.

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# Original Communications

## THE PERIVASCULAR SPACE OF THE PULMONARY VESSELS

### An Anatomic Demonstration*

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It is generally conceded that a cleavage plane exists around the pulmonary vessels. Under certain pathologic conditions, this potential perivascular space may be converted into a very real and actual space. Escape of air in several clinical conditions, such as pulmonary interstitial emphysema, into and along these perivascular channels from alveoli which are based along its margins has been postulated and demonstrated by several investigators.[1, 2] Macklin[2] reported the observation of air bubbles in the pulmonic arterial and venous vascular sheaths following intratracheal introduction of air under pressure into the lungs, and noted that the air often found its way into the opposite lung, as well as into the mediastinum. Similar observations are reported by Joannides and Tsoulos.[1] In the isolation of pulmonary blood vessels for ligation, limited sectional demonstrations of this cleavage plane are easily exhibited. However, although textbooks generally mention the existence of a fibrous sheath enclosing vessels, actual anatomic demonstration of this space has been lacking, and there is a paucity of information concerning the peripheral and central extent and relations of this cleavage plane.

We have endeavored to demonstrate this pulmonary perivascular space in the dog by gross, microscopic, and roentgenographic methods in order to determine the relations of this space to the vascular trunks and arborizations within the lung as well as its central relations to the pericardium.

## METHOD

The perivascular space was entered at the hilum by blunt dissection through as small an opening as possible, and identified by the fact that not until the perivascular space was entered did separation along a cleavage plane by blunt dissection occur with ease.

A small length of polyethylene tubing of a diameter sufficient to permit a blunted No. 18 needle to fit very snugly into its lumen at one end was introduced into the perivascular space. The tube was secured by placing a ligature tightly around the vessel, compressing the tubing between the outer wall of the perivascular space and the wall of the enclosed vessel

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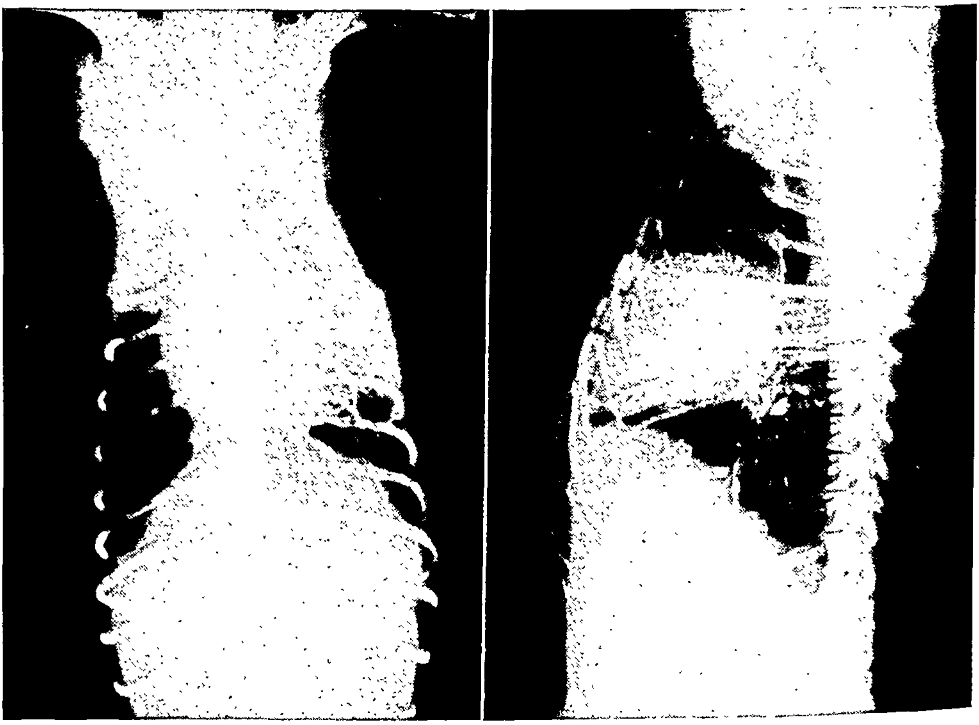
*An exhibit of this anatomic demonstration of the pulmonary perivascular system was presented at the meeting of The American Association of Anatomists in Detroit, Mich., in March, 1951.

in such a manner as to exclude the opening through which the tubing had been introduced. Ten to 20 per cent bismuth oxychloride (the less concentrated solution being used in the living animals) was injected under moderate pressure through the polyethylene tube into the closed space thus created.

While a tubing of smaller diameter might have been preferable, the injected radiopaque solution was forced through even the No. 18 needle with difficulty, a 50 c.c. syringe being used to permit the application of moderate force. The nature of the bismuth oxychloride solution made it possible to observe the course of the injection grossly as it dissected its way along the perivascular space, and secure roentgenographic demonstrations of the extent and pattern of its spread, as well as microscopic sections showing the relation of the injected material to the lung structures and vessel wall.



*A.* *B.*

Fig. 1.—Anteroposterior and lateral roentgenograms of a living dog taken five days after left unilateral injection of pulmonary artery demonstrating ipsilateral and contralateral periarterial spaces.

In heart-lung preparations removed en masse, and in living animals in which peripheral and central injections were made, the ligature was first passed through a small rubber ball approximately 0.5 cm. in diameter, which was then placed behind the vessel after the artery or vein had been bluntly dissected free of the surrounding tissues, care being taken to stay well outside of the perivascular space. The rubber ball, upon tying this ligature, compressed the tissues without harming the vessel wall, and could easily be released to permit redirecting of the tubing in the perivascular space into the opposite direction for further injection. Due to mechanical difficulties involved in some of the injections, particularly in those of the veins of individual lung specimens, the larger tributaries rather than the main pulmonary vessels were injected individually.

Injection was continued in each direction until the pressure of the fluid in the perivascular space caused marked bulging of the surrounding wall, until leakage occurred, or until the injected solution could be seen well out around the small vessels in the periphery of the lung.

Using individual right- and left-lung specimens, injections were made to demonstrate the following perivascular spaces peripherally on each side: (1) inferior pulmonary vein, (2) superior pulmonary vein, (3) inferior and superior pulmonary veins, (4) pulmonary artery, and (5) all vessels.

Using individual heart-lung preparations removed intact en masse, injections were made to demonstrate the perivascular spaces peripherally and centrally as follows: (1) left inferior pulmonary vein, (2) left superior pulmonary vein, (3) left pulmonary artery, (4) right inferior pulmonary vein, (5) right superior pulmonary vein, and (6) right pulmonary artery.

Using living animals, injections were made to demonstrate the perivascular spaces as follows: (Dog No. 1) left inferior pulmonary vein, peripherally; (Dog No. 2) left superior pulmonary vein, peripherally; (Dog No. 3) left inferior and superior pulmonary veins, peripherally; (Dogs No. 4 and No. 5) left inferior pulmonary vein, peripherally and centrally; (Dogs No. 6 and No. 7) left superior pulmonary vein, peripherally and centrally; (Dogs No. 8, No. 9, No. 10, No. 11, No. 12, and No. 13) left pulmonary artery, peripherally and centrally.

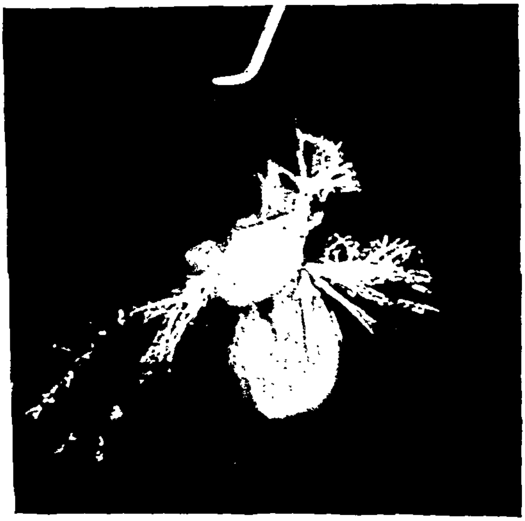


Fig. 2.—Peripheral and central injection of inferior pulmonary vein at point marked by arrow, demonstrating contralateral inferior pulmonary vein and ipsilateral superior pulmonary vein.

As a corollary study, the lungs of four heart-lung specimens were inflated by intratracheal insufflation under pressure. In two specimens in which air appeared in the perivascular spaces and traveled centrally into the hilar region, the space created by the passage of air along the vessels was injected with bismuth oxychloride solution and studied microscopically.

## EXPERIMENTAL OBSERVATIONS

All peripheral injections were made with comparative ease. Roentgenograms taken with the lungs inflated revealed the existence of a continuous cleavage plane around the most distal branches and tributaries of all the pulmonary vessels (Figs. 1 and 2). Microscopic sections of the injected tissues revealed the injections to have entered a discrete perivascular space, closely following the most minute branchings of the vessels (Figs. 3, 4, and 5).

Injections directed centrally into the arterial perivascular space of two living dogs resulted in the solution breaking into the mediastinum. However, in two of the dogs the perivascular space of the main pulmonary artery as well as that of the contralateral vessel was clearly shown on roentgenograms taken immediately and several days after injection (Fig. 1). Similar findings resulted from the injections around the arteries of the heart-lung preparations. Careful examination revealed no evidence of the injected solution within the blood vessels.

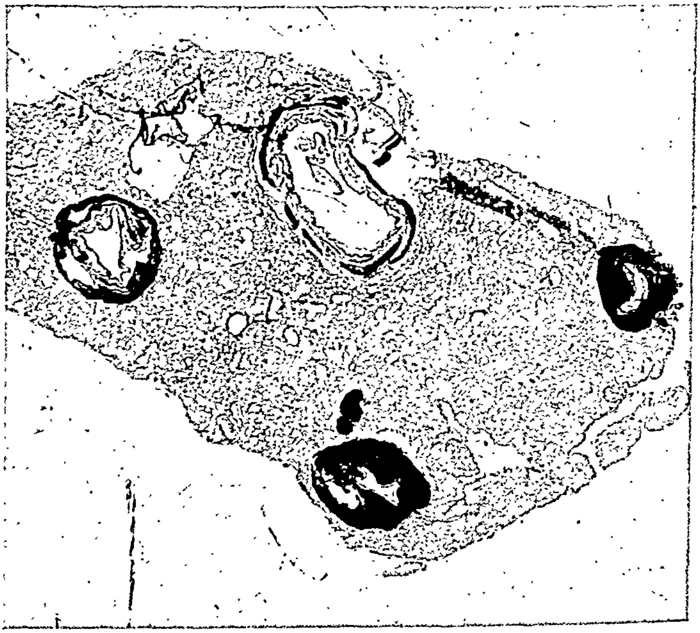


Fig 3.—Low-power field demonstrating bismuth mass in perivascular spaces.

Injections centrally into the perivascular space of the pulmonary veins were consistently unsuccessful in the living animals. In the heart-lung preparations these injections, occasionally accomplished with comparative ease, often required repeated trials. With the pericardial sac opened, it was noted that the injections often resulted in the solution breaking through the serous pericardial covering of the left atrium at several points. By plugging these several leaks as they occurred by placing pressure over this region with our fingers, the solution could be forced beyond and eventually reached the contralateral perivascular spaces, at which time there was a distinct decrease in the resistance to the injection. Central injection of the perivascular space of one pulmonary vein in several specimens resulted in the spread of the solution to the other ipsilateral vein as well as to the contralateral veins (Fig. 2).

Microscopic slides made following injection of the perivascular space created by the passage of air during intratracheal insufflation under pressure of two heart-lung preparations showed the solution to be in a perivascular space similar in all respects to those studied by the above methods.

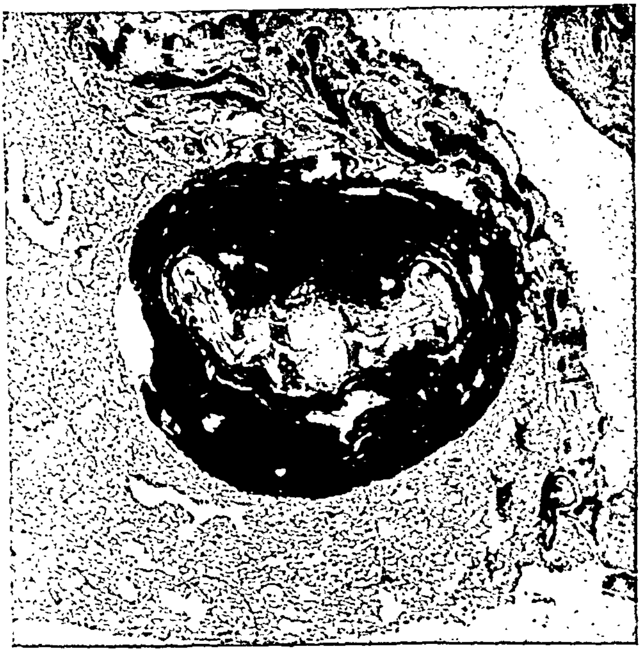


Fig. 4.—Periarterial injection.

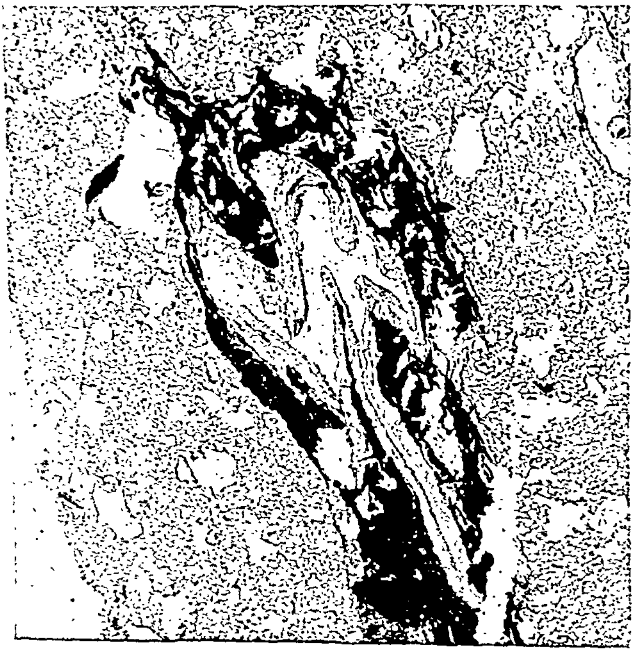


Fig. 5.—Perivascular space demonstration at vascular bifurcations.

## DISCUSSION

By peripheral and central injection of a radiopaque solution under pressure into the cleavage plane between the pulmonary vessel walls and the surrounding tissues at the hilum of the dog's lung in the living animal, as well as in individual lung specimens and in heart-lung preparations removed en masse, definite dissectable ipsilateral and corresponding contralateral perivascular spaces have been identified grossly, microscopically and roentgenographically.

The injected solution apparently follows the line of least resistance in spreading along this areolar cleavage plane. Embryologically, the pulmonary veins have as an adventitia a very loose mesenchymal structure whose elements are largely longitudinal and which may be very easily split under even slight injection pressure for a distance which, peripherally, is conjectural, and it has been suggested that the injected solution follows such a plane.[3] Within the lung, the bases of the marginal alveoli appear to make a tunnel-like investment for arteries and veins, enclosing the perivascular space between the vessel walls and the marginal alveolar bases. In the case of the arteries, it has been suggested that the injected solution is guided by the adventitial covering of the vessels peripherally and centrally.

With regard to the injection of the contralateral venous perivascular spaces, the possibility that the solution finds its way across the midline into the corresponding perivascular space by chance is to be considered. However, it is speculated that at the hilum this perivascular sheath represents an extension of the fibrous pericardium along the vessels. The pericardial space itself is apparently separated from the perivascular space by the reflection of the serous pericardium. The reflection of the pericardium at the junction of the pulmonary veins with the right atrium suggests a continuous nature for this space across the midline connecting the perivascular spaces of all the pulmonary veins as well as, probably, those of the superior and inferior vena cavae.[4]

From practical experience in the performance of pulmonary resections, exposure of the vessels at the hilum as well as in the fissures is greatly facilitated by dissecting within this areolar perivascular space, which is relatively avascular and has been found to remain surprisingly free even in cases exhibiting advanced lymphadenopathy, excepting, of course, those cases showing advanced calcification or carcinomatous infiltration.

Once this perivascular space is entered and the vessel freed within its tunnel, clean incision of any overlying structures can be practiced with safety. This will often save much in time as well as offer freedom from annoying bleeding.

## SUMMARY

The existence of a readily identifiable perivascular space about the pulmonary vessels has been anatomically demonstrated by gross, microscopic and roentgenographic methods, showing the relations of this space to the vascular trunks and arborizations within the lung. Its relation to the pericardium and the contralateral vascular bed has been commented upon.

The information acquired concerning the nature and extent of these perivascular spaces is of great practical value in performing pulmonary resections in clinical cases, and may well explain pathologic processes in several clinical conditions.

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# CYSTIC DISEASE OF THE LUNGS

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THE incidence of cystic disease of the lungs is greater than earlier reports would appear to indicate. Although pulmonary cysts have long been recognized as a pathologic entity, the diagnosis was infrequently made until Koontz[1] introduced the subject into American literature in 1925. Eleven years later Schenck[2] was able to find only 233 cases in the literature, two-thirds of which were in foreign publications. Since that time the importance of this disease has become well recognized, as is evidenced by the now voluminous literature on the subject. The incidence, however, has been difficult to ascertain, principally because of differences in classification and variations in nomenclature. Hospital dispositions and routine autopsy reports are recognized sources of information on disease incidence. At Walter Reed Army Hospital there were 51,199 dispositions during the period from February, 1947, to January, 1951. Of this group there were twenty-one patients in whom the diagnosis of congenital bronchogenic cysts was made. The incidence based on these data is forty-two cases of bronchogenic cysts per 100,000 hospital dispositions. Other pulmonary cysts such as subpleural blebs and bullous emphysema could not be traced because of improper classification. However, during this period there were fourteen cases of recurrent pneumothorax that were caused by rupture of subpleural blebs treated surgically.[3] Weaver and von Hamm[4] reported two cases of pulmonary cysts in 6,000 consecutive autopsies, although Lederer[5] found none in 5,000 autopsies, many of which were performed on infants and children. The advent of routine chest roentgenograms has been another source of information in the establishment of the incidence of this disease. Edwards and Ehrlich[6] found two cases of pulmonary cysts and one case of bullous emphysema in preinduction chest roentgenograms of 41,819 inductees and guardsmen. From their figures it can be estimated that the incidence of congenital cystic disease of the lungs in asymptomatic young adults is five per 100,000 population.

It is obvious from the differences in the above figures that neither routine roentgenograms, autopsy specimens, nor hospital dispositions alone give an accurate picture of the incidence of cystic disease of the lungs. This is particularly true of x-ray examinations when it is realized that routine chest roentgenograms will not reveal all types of pulmonary cysts. Two of the cases reported in this series could not have been diagnosed by routine chest roentgenograms.

It is even more difficult to determine the incidence of pulmonary blebs and bullae; this is due in a large measure to the lack of uniformity in diagnosis and in most instances the lack of pathologic specimens to confirm the clinical impression. However, it can be stated with some conviction that cystic disease of

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the lungs is a relatively common disease and that the diagnosis must be considered in any case in which chest symptoms persist and in which the diagnosis is in doubt.

There have been so many articles in the recent medical literature on cystic disease of the lungs that the authors hesitate to add more to what is already an overcrowded field. Despite the extensive literature on this subject, there is still much confusion as to the classification and nomenclature of this disease. Mooreman,[7] in the chapter on pulmonary cysts in *The Chest and the Heart,* makes this statement: "Increasing knowledge has not only failed to simplify diagnosis but it has confused rather than clarified the question of classification." This state of confusion is shared by other authors. In the 1947 edition of Boyd's[8] *Surgical Pathology,* this statement is made: "It is not possible to write a satisfactory account of cysts of the lung for the pathology is so confused and there is no correlation between the pathological and clinical picture." The varied nomenclature has indeed served to becloud the issues. At the Armed Forces Institute of Pathology[9] in Washington, D. C., there are twenty-eight synonyms under which cases of cystic disease of the lung are filed (Table I).

TABLE I

| | |
|---|---|
| Congenital bronchiectasis | 18 |
| Fetal bronchiectasis | 1 |
| Embryonic cyst of the lung | 1 |
| Cyst, multiple, lung, congenital | 3 |
| Cystic bronchiectasis | 12 |
| Polycystic disease, congenital | 2 |
| Multiple pulmonary cysts | 15 |
| Bronchogenic cysts | 99 |
| Emphysema, congenital | 1 |
| Adenoma, bronchogenic, congenital | 1 |
| Cyst, bronchial, congenital | 6 |
| Cyst, solitary, congenital, pulmonary | 60 |
| Bronchiectasis, bronchiolar | 1 |
| Cysts, inclusion, multiple, lung | 1 |
| Cystlike cavity, lung | 1 |

TABLE II

- Bronchiectasis neonatorum
- Atelectatic bronchiectasis
- Telangiectasis
- Bronchiolectasis
- Cystic fetal bronchial adenoma
- Honeycomb lung
- Congenital pulmonary lymphangiectasis
- Congenital cystic malformation of the lung
- Cystic inclusion of the lung

Other synonyms have been gleaned from the literature (Table II). Many of the diagnoses listed here were used by earlier writers and were based on the then current concepts of the etiology of the disease. Koontz,[1] and later Schenck,[2] in a clinicopathologic study of cystic disease of the lung, reported in detail the various theories of pathogenesis which have at one time or another been proposed for congenital cysts of the lung. Many of these older terms have been discarded and the theories of pathogenesis discredited. However, there are still a host

TABLE III. CYSTIC DISEASE OF THE LUNG

- I. Congenital Pulmonary Cysts.
  - A. Bronchogenic cell type
    - 1. Solitary
    - 2. Multiple
  - B. Alveolar cell type
    - 1. Solitary (balloon cyst, pneumatocele)
    - 2. Multiple
  - C. Bronchogenic and alveolar types combined
- II. Acquired Pulmonary Cysts.
  - A. Bullous emphysema
  - B. Subpleural blebs

of pathologic conditions erroneously classified under cystic disease of the lung, and there are also many cases of cystic lung disease which are not recognized and therefore improperly classified. In recent years we have used a classification which includes all types of cystic lung disease, and which we believe will be acceptable to both clinician and pathologist. It is not the purpose of the authors to add to the nomenclature already exhaustive in variety and scope. On the contrary, this classification utilizes the terminology which is most frequently found in present-day literature (Table III).

For the purpose of classification, cystic disease of the lungs can be divided into two large groups: the congenital and the acquired.[10-12] The acquired type of so-called cystic lung disease should probably be included under emphysema and not in this classification. However, because most authors include acquired as well as congenital cysts in their series, and also because it is occasionally not possible to distinguish between the two, both types of cystic disease will be discussed as a group.

Chronic lung abscess has not been included in this classification. Although such cavitations are sometimes termed acquired cysts or abscess cysts, the justification for such terminology being that in some chronic pulmonary abscesses, there is an ingrowth into the cavity of cuboidal or columnar epithelium.[13] Although it is a well-recognized fact that squamous metaplasia frequently occurs in such cases, ciliated epithelial replacement of the pyogenic membrane of an abscessed cavity occurs so rarely, if it occurs at all, that the authors have purposely not included that type of case. The term chronic lung abscess defines so clearly that pathologic entity that an attempt to include it in a classification of pulmonary cysts would in our opinion be a mistake.

## CONGENITAL PULMONARY CYSTS

Congenital cysts of the lung are one of three types, depending upon the cellular component of the cyst wall. The bronchogenic cysts are those in which the wall of the cyst is lined by bronchial mucosa with either tall columnar or cuboidal ciliated cells. These cysts may be single or multiple. They may be confined to a segment or diffusely scattered throughout the entire lung. Small, diffusely scattered cysts which communicate with the bronchi have the appearance on bronchography of saccular bronchiectasis. When seen in infancy or childhood, this type case has frequently been classified as congenital bronchiectasis, fetal bronchiectasis, or bronchiectasis neonatorum. Superimposed infection will in many instances make a proper diagnosis difficult, particularly if the infection is long standing. There are certain features in which the congenital malformation differs from acquired bronchiectasis. The distribution of the lesion is an important indication of the nature of the disease. In acquired bronchiectasis the dependent portions of the lung, particularly the basilar segments of the lower lobes and lingular segment of the upper lobe, are most frequently involved. In congenital cystic disease of the small, diffuse type, the upper lobes are often the site of the lesion. In the congenitally malformed lung the cystic areas in many instances are of a uniform size compared to both tubular and saccular dilatations seen in acquired bronchiectasis. The histologic picture in the congenital malformation will characteristically show bronchial type mucosa unless superimposed infection has destroyed this membrane. In the acquired disease in which sacculations are apparent, there will be evidence of considerable inflammation and scarring. Two cases of congenital cystic disease of the lung have recently been reported by Ravitch and Hardy[14] in which they described the bronchographic appearance of the cysts as "grapes hanging from a stem."

As pointed out by Anspach and Wolman,[15] the postnatal behavior of congenital cysts will depend in a large degree upon whether or not they communicate with a bronchus. Although a bronchial communication may not be present, infection may enter the cyst by extension from adjacent diseased tissue. In the authors' experience all infected cysts have communicated with the airway; whether the infection preceded the bronchial communication or not is not known. The converse that all cysts with bronchial communications are infected is, of course, not true.

The large solitary cysts of bronchogenic origin are frequently found projecting from the mediastinum. Some of these cysts have on occasion been classified as accessory lobes or accessory lungs.[16] DeBakey[17] considers the appearance of normally developed lung parenchyma about the periphery of the bronchogenic cyst sufficient reason to classify these malformations as accessory lobes despite the fact that the vascular pedicle arises from the systemic rather than the pulmonary circulation. In the classification proposed here mediastinal cysts of bronchogenic origin have purposely not been included.

The alveolar type cysts are those in which the wall of the cyst is composed of flat squamous cells characteristic of the air sacs.[18] This type of cyst always has a bronchial communication. Rarely, as a result of infection, these cysts may contain both fluid and air. The majority, however, are air cysts and produce symptoms by encroaching on normal lung tissue. Spontaneous cures have occurred,[2, 19, 20, 21] and such a case will be reported in this paper. Spontaneous cures may occur as a result of closure of the bronchial communications and resorption of the air with obliteration of the cyst. In the case of this type reported by Lloyd,[19] the period of follow-up covers ten years. Although the cyst he described occurred in a small child and appeared to be a solitary cyst, it was classified as a case of bullous emphysema.

Benjamin and Childe[22] reported a series of nineteen cases of "Localized Bullous Emphysema Associated with Pneumonia" in children from ages 3 months to 13 years. In a similar article entitled "Regional Obstructive Emphysema in Infants and Children," Coffey[23] reported on twenty such cases. All of the patients in both series underwent spontaneous cure. The cases illustrated in those articles are similar to the type cases which we have classified as congenital in origin. Although we are not convinced that such a stand on our part is entirely correct, there are certain facts which must be considered. Pneumonic infiltration is a relatively frequent complication of cystic lung disease, and it is possible in some of their illustrated cases that the pulmonary cysts were complicated by pneumonia rather than cystic areas resulting from the pneumonia. In addition, bullous emphysema as we know it in the adult runs a clinical course so entirely different from the cases reported by Benjamin and Childe and by Coffey that one hesitates to accept the two pathologic conditions as similar. If in the course of future investigation it is demonstrated that this disease is bullous or obstructive emphysema, then it will be necessary to divide bullous emphysema into an adult and an infant type because of the extreme difference in the clinical course of the two disease processes. At the present time we are manifestly handicapped because there have not been specimens for pathologic examination.

The large solitary balloon cysts or pneumatoceles have been included under congenital cysts for several reasons. These cysts occur more frequently in infants and children.[3, 12, 24] The structure of the solitary cysts is quite different from the emphysematous bullae[25] from which they must be differentiated. The solitary cyst is completely lined with alveolar type cells and as a rule has a single bronchial communication. All adjacent pulmonary segments are anatomically normal except for compression atelectasis due to the enlarging balloon cyst. The fact that these cysts have been found in adults does not necessarily refute the claim that they are congenital in origin. The important point is not whether these cysts are congenital in origin or not, but because of the difference in prognosis and management, they must be distinguished from emphysematous bullae.

The congenital cysts in which there exist both bronchogenic mucosa and the flat alveolar cells of the air sacs are rare, but have been reported.

Congenital cystic disease of the lung results from an anomalous development of the lung anlage, and the characteristic cellular components of the malformation will be determined by the age of the individual at the time that the malformation occurs. If Broman's[26] observations are correct, the malformations could conceivably occur after birth and may account for some of the bizarre clinical manifestations seen in infants and children. He quotes the work of von Selenka, Narath, and Brehmer to corroborate his observations. Broman states ". . . that the definitive lung parenchyma (total alveolar mass of the adult) of man and the other placentalia, for the most part becomes built after birth." He further states that ". . . entirely new alveoli and alveoli processes develop after birth." This process is complete and adult type lung parenchyma is established in early adolescence.

The following case reports will be used to illustrate the proposed classifications. The authors are indebted to Colonel Joe M. Blumberg, M.C., Pathologist, Walter Reed Army Hospital, and Dr. Theodore Winship, Consultant in Pathology, Walter Reed Army Hospital for reviewing with us the pathology in each case. Some of the problems which arise in the management of cystic lung disease will be discussed.

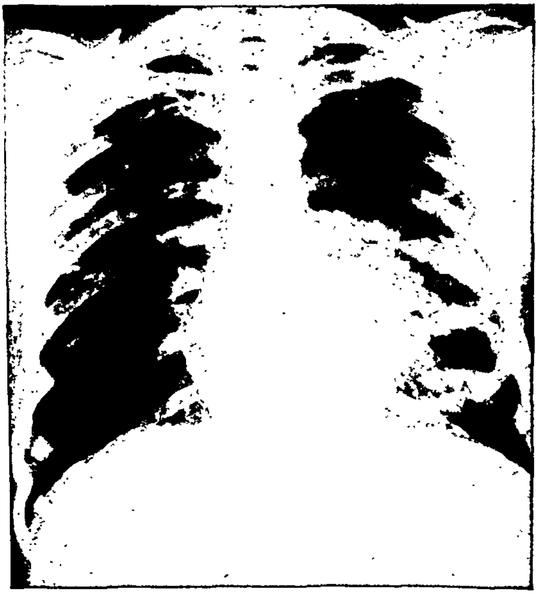

Fig. 1.—(Case 1.) Posteroanterior roentgenogram of the chest showing multiple radiolucent areas in the left lower lung field.

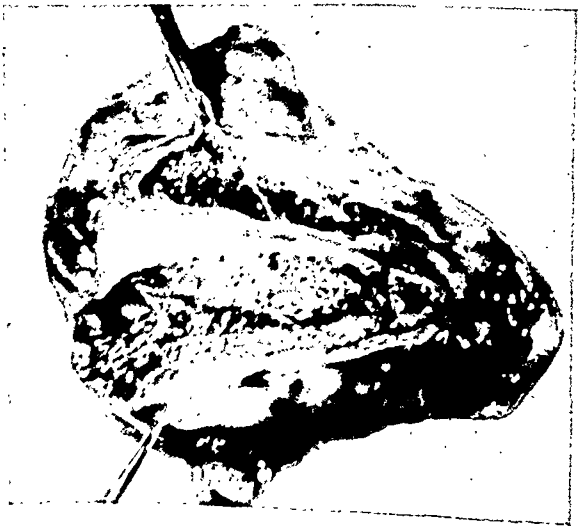

Fig. 2.—(Case 1.) Specimen showing infected bronchogenic type cysts.

Case 1.—A 23-year-old white man was admitted to the hospital with chest pain, productive cough, and fever of six weeks' duration. During the nine months prior to admission, the patient had had several similar episodes. Each episode which was diagnosed as pneumonia had responded slowly to antibiotics. A chest roentgenogram showed multiple radiolucent areas in the left lower lung field (Fig. 1), in one of which there is a fluid level. Bronchograms showed no filling of the superior segment of the left lower lobe and one large saccule in the posterior basilar segment. A diagnosis of cystic disease

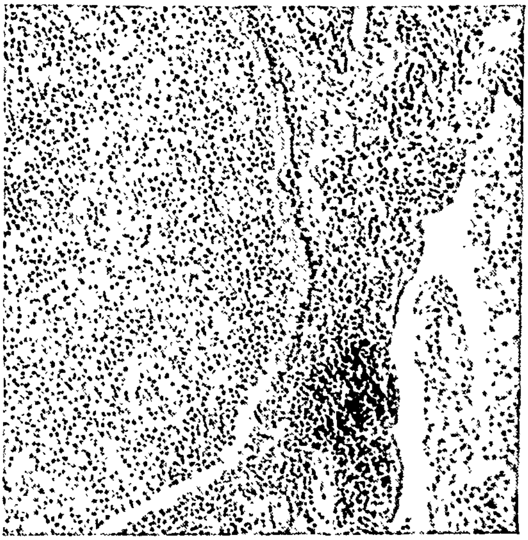

Fig. 3.—(Case 1.) Photomicrograph showing the ciliated columnar epithelium. Neutrophilic granulocytes can be seen in the exudate.

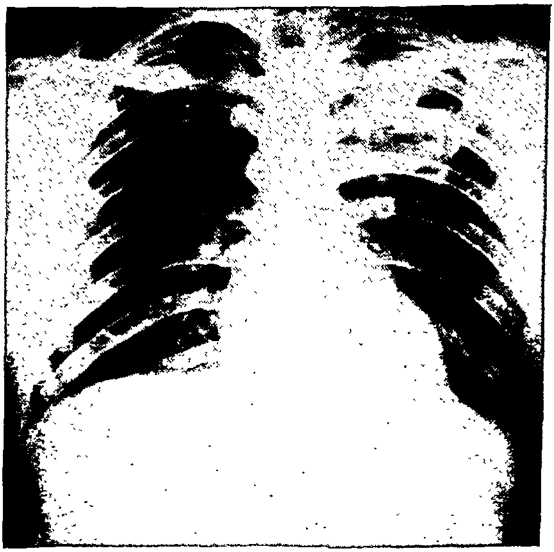

Fig. 4.—(Case 2.) Posteroanterior roentgenogram of the chest showing multiple radiolucent areas in the left upper lobe.

of the lung confined to the left lower lobe was made and a left lower lobe lobectomy was performed. The specimen revealed many cystic areas in the superior and posterior basilar segments of the left lower lobe which were filled with pus and showed evidence of considerable inflammatory reaction involving the contiguous lung tissue (Fig. 2). The cyst wall on the microphotograph shows the tall, columnar ciliated bronchial epithelium (Fig. 3). Neutrophilic granulocytes are seen in the exudate. This case is classified as congenital cystic disease of the lung, bronchogenic type, multiple cysts confined to the left lower lobe.

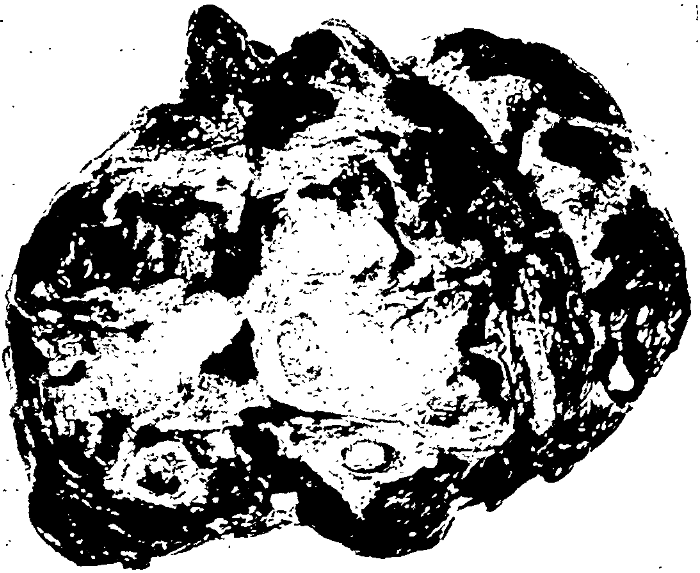

Fig. 5.—(Case 2.) The specimen showing large multiple cysts involving the apical posterior segment of the left upper lobe.

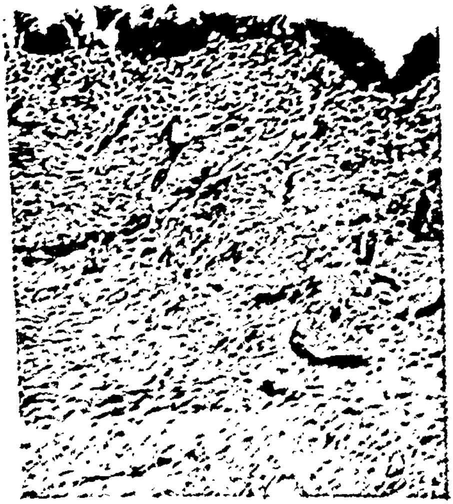

Fig. 6.—(Case 2.) Photomicrograph of the cyst wall showing tall columnar, ciliated epithelium.

CASE 2.—This 31-year-old white man was admitted with the following history: six months prior to admission he complained of pain in the chest, became febrile, and coughed up large amounts of pus. A roentgenogram at that time revealed radiolucent areas in the left upper lobe (Fig. 4). Later roentgenograms of the chest revealed decrease in the inflammatory reaction in the left apex, but the radiolucent areas were still apparent. The patient became asymptomatic. Bronchograms revealed a normal tracheobronchial tree on the left except for absence of filling of the apical posterior segment of the upper lobe. Tuberculosis work-up was entirely negative. At operation cystic disease confined to the apical posterior segment was removed by segmental resection. The patient made an uneventful convalescence and returned to duty.

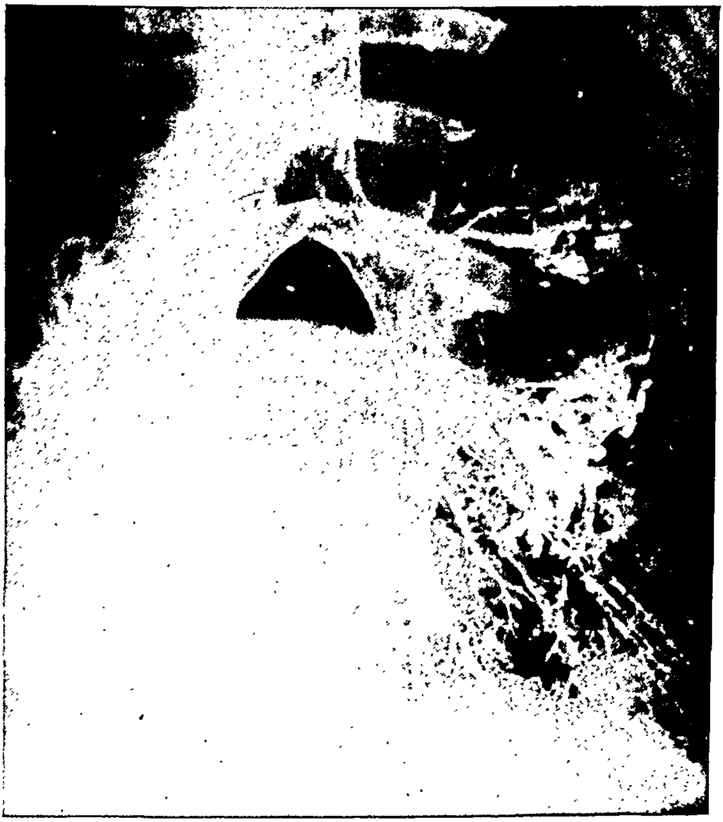

Fig. 7.—(Case 3.) Bronchography showing what was believed to be saccular bronchiectasis involving the right upper and right middle lobes.

*Pathology:* Numerous fairly large cysts were demonstrated (Fig. 5). Tall, columnar ciliated epithelium lined the cyst walls (Fig. 6). This case is also classified as congenital cystic disease of the lung, bronchogenic type, multiple cysts, confined to the apical posterior segment of the left upper lobe.

*Comment.*—These two cases represent fairly large bronchogenic cysts which were found deep in the structure of the lung. The symptoms were caused by secondary infection. The cysts communicated at least periodically with the airway which allowed for drainage of their contents. Repeated respiratory infections with productive cough and fever and persistence of abnormal roentgenograms of the chest brought these patients to the hospital.

CASE 3.—This 18-year-old white man gave the history of an episode of chest pain and hemoptysis at the age of 14 years following a tonsillectomy. At that time he was told that he had a lung abscess which drained beneath the right axilla. This drained periodically for two years. Prior to enlistment he had been asymptomatic for two years. Soon after basic training, however, he had an upper respiratory infection following which he noticed purulent drainage and air escaping from the old sinus tract in his right axilla. Bronchography revealed what was considered saccular bronchiectasis involving the right upper and right middle lobes (Fig. 7). The sinus tract communicated with a large bronchus in the right middle lobe. A right upper and middle lobectomy was completed. The operation was staged because of anesthetic difficulties. The patient made an uneventful recovery and was returned to military duty.

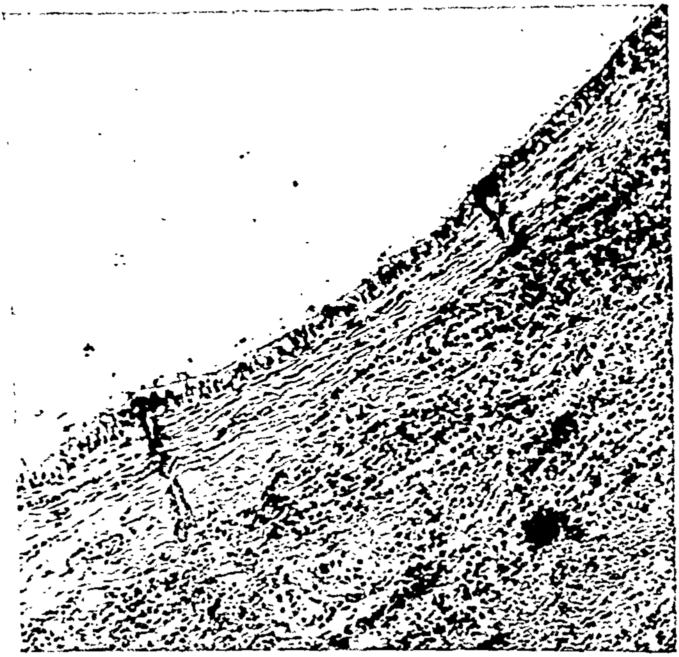

Fig. 8.—(Case 3.) Photomicrograph of a section taken through one of the cystic areas showing ciliated bronchogenic mucosa lining the cyst.

Pathologic examination showed small cystic areas which communicated with the small bronchi. These were diffuse throughout the entire upper and middle lobes. Microscopic examination of the tissue revealed the characteristic cellular pattern of congenital bronchogenic cysts (Fig. 8).

*Comment.*—The distribution of the cystic areas as seen on bronchograms, namely, that the disease was completely confined to the middle and upper lobes, plus the fact that the sacculations seemed fairly uniform in size with no tubular dilatations, suggested the possibility of congenital malformations within the

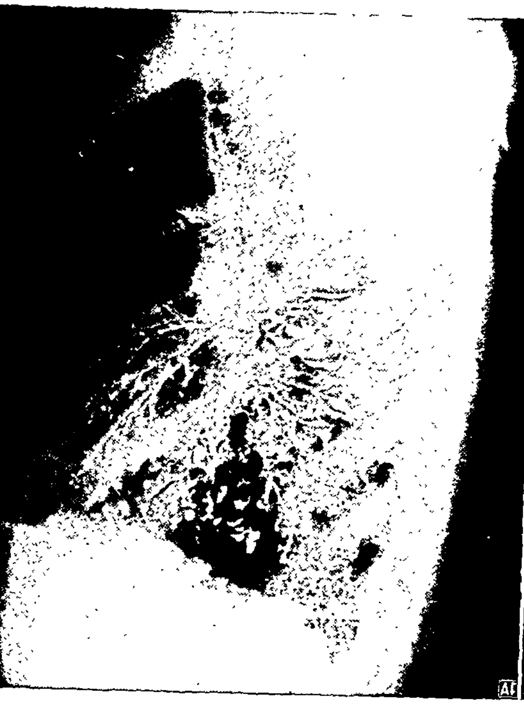

Fig. 9.

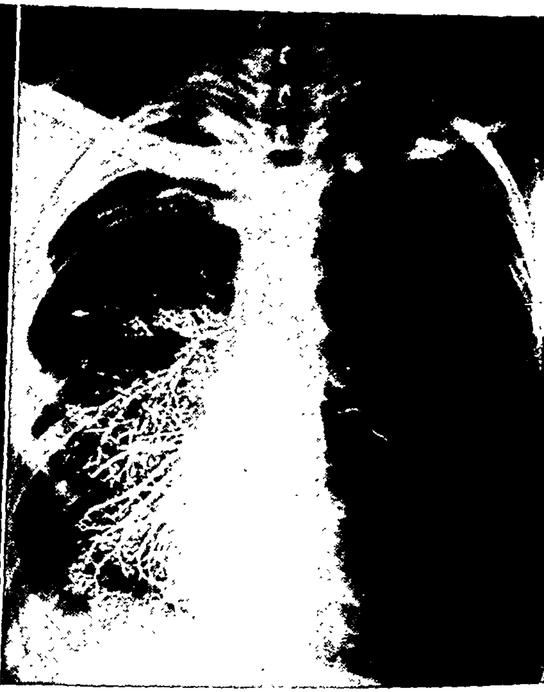

Fig. 10.

Fig. 9.—(Case 4.) Bronchography demonstrating saccular bronchiectatic dilatations involving the right lower and posterior segment of the right upper lobe.

Fig. 10.—(Case 4.) Postoperative bronchogram showing good expansion of the remaining pulmonary segments.

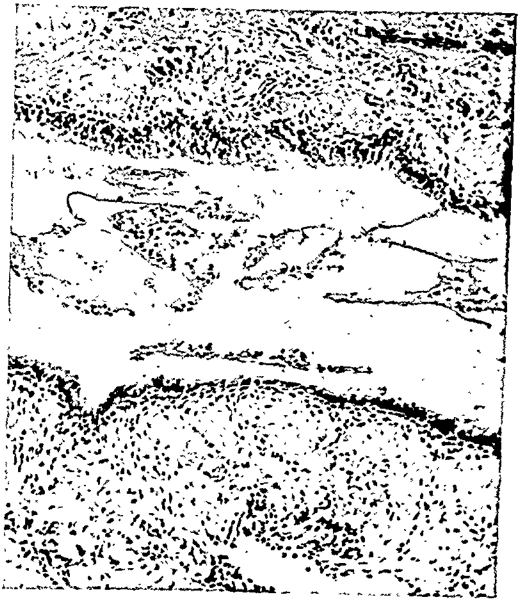

Fig. 11.—(Case 4.) Photomicrograph of a section through one of the cystic areas demonstrating bronchogenic type mucosa characteristic of congenital pulmonary cysts.

pulmonary parenchyma. Careful examination of the specimen by the pathologist confirmed the clinical diagnosis of congenital cystic disease of the lung, bronchogenic type, multiple, confined to the right upper and right middle lobes.

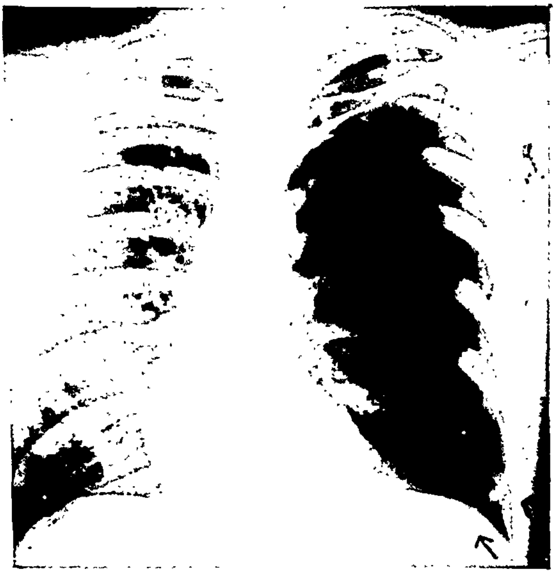

Fig. 12.—(Case 5.) Posteroanterior roentgenogram of the chest showing lack of lung markings and almost complete replacement of lung tissue of the left hemithorax. A small pneumothorax exists in the right lower lung field.

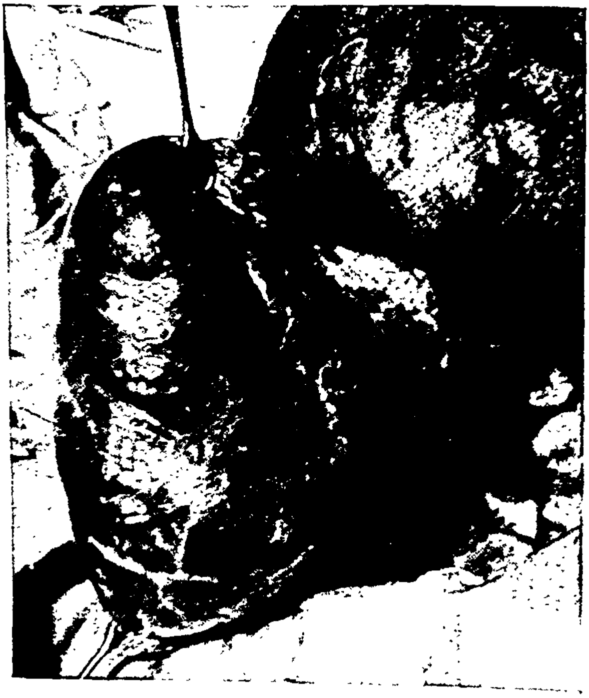

Fig. 13.—(Case 5.) Specimen at operation.

Case 4.—This 21-year-old white man presented symptoms of a chronic productive cough of four years' duration. He gave a history of having had bronchopneumonia at four months of age with several recurrences of pneumonia during infancy. Bronchography revealed saccular bronchiectatic dilatations involving the entire right lower lobe and the posterior segment of the right upper lobe (Fig. 9). The patient was operated on, and a right lower lobectomy and segmental resection of the posterior segment of the right upper lobe was accomplished. The patient recovered and returned to duty. Postoperative roentgenogram of the chest revealed good expansion of the remaining pulmonary segments (Fig. 10).

The pathology report was of multiple small cysts lined with bronchial type epithelium involving the entire right lower lobe and the posterior segment of the right upper lobe (Fig. 11). These cystic cavitations communicated with the bronchi. This case is classified as congenital cystic disease of the lungs, bronchogenic type, multiple, confined to the right lower lobe and posterior segment of the right upper lobe.

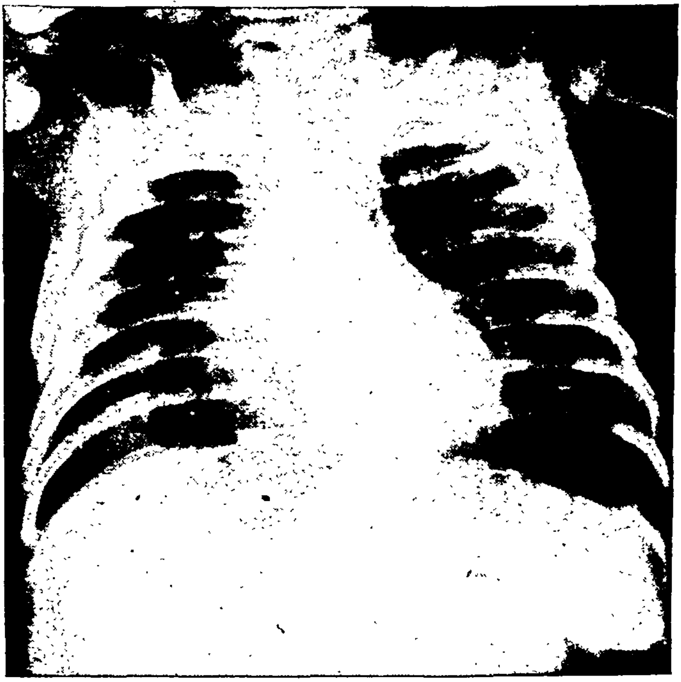

Fig. 14.—(Case 6.) Posteroanterior roentgenogram of the chest showing multiple cystic areas in both lung fields.

*Comment.*—This case represents congenital cystic disease of the lungs of the bronchogenic type which could easily have been confused with saccular bronchiectasis. The bronchographic findings of saccular dilatations of uniform size plus the diffuse distribution throughout the lower lobe and the posterior segment of the right upper lobe was the first clue that this was cystic disease and not acquired bronchiectasis. Pathologic examination which included a very careful inspection of the gross specimen showed the cystic areas to be lined with the characteristic columnar ciliated cells.

Case 5.—This 27-year-old white man was admitted to the hospital with a diagnosis of pneumothorax. The patient was asymptomatic. Roentgenograms of the chest which accompanied him showed complete lack of lung markings in the left hemithorax except for a few fine shadows about the pulmonary hilum. One film, however, which had been taken following thoracentesis showed air in the free pleural space and the wall of a cyst well outlined (Fig. 12). Operation was performed and a giant, solitary air cyst was removed (Fig. 13). The patient recovered and was sent to duty.

*Pathologic examination:* A large, solitary pulmonary cyst was seen, the walls of which were lined with flattened fusiform cells. This was classified as congenital cyst of the lung, solitary alveolar cell type.

*Comment.*—This type cyst represents a large, solitary, balloon cyst or pneumatocele which is most frequently found in infants and children. It is very thin walled and is often mistaken for a pneumothorax, as happened in this case. This cyst is classified as congenital. It should not be confused with emphysematous bullae.

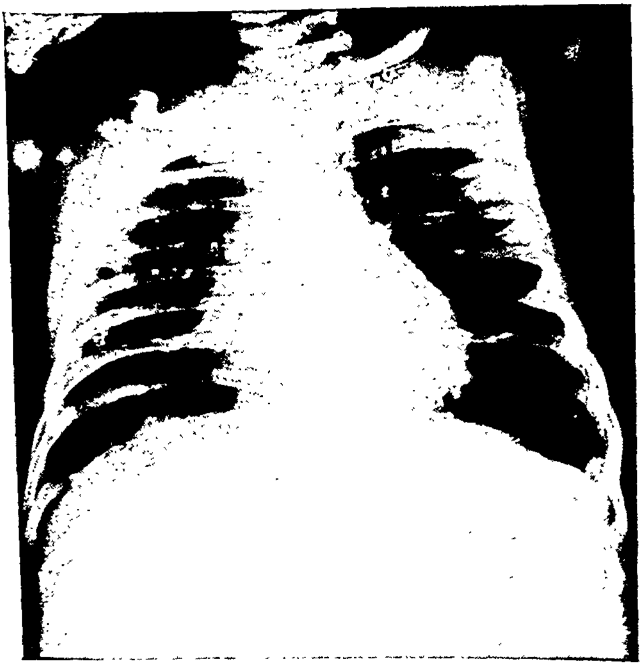

Fig. 15.—(Case 6.) Posteroanterior roentgenograms of the chest ten days later, showing increase in the number of cystic areas within the lung fields.

CASE 6.—This 5-week-old, normally delivered, Negro girl developed a severe respiratory infection with dyspnea and abdominal distention several days prior to admission. X-ray examination of the chest revealed several cystic areas in the lower lung fields, bilaterally (Fig. 14). The child improved symptomatically under treatment, but repeat x-ray examination showed enlargement of the cystic areas and an increase in the number of cysts until both lower lobes seemed to be diffusely involved (Fig. 15). The child improved with antibiotic therapy and within a week x-ray examination showed diminution in the size of the cysts. A diagnosis of congenital cystic disease of the lungs was made at that time. She was discharged from the hospital at the age of 7 weeks. During the following nine-month period she had several bouts of respiratory infections with cough and slight to moderate dyspnea. In each instance she improved remarkably with treatment. The last several respiratory infections were notable in that dyspnea was absent. At the age of 10 *months roentgenograms of the chest showed no evidence of disease* (Fig. 16). Bronchograms adequately filled all pulmonary segments with no evidence of any cystic areas or bronchiectatic dilatations.

*Comment.*—This child has apparently undergone a spontaneous cure. Spontaneous cures of lung cysts have been reported.[19-23] Because of the age of the patient and the course of the disease, it is assumed that these cysts were congenital in origin. This case has been classified as congenital cystic disease of the lungs, alveolar cell type.

*Fig.* 16.—(Case 6.) Posteroanterior roentgenograms of the chest seven months following initial x-ray film showing a completely normal chest to this type of examination.

### ACQUIRED CYSTIC DISEASE OF THE LUNGS

Emphysematous bullae are the result of a breakdown of the interalveolar septa with the formation of cystic areas within the lung parenchyma. As this process continues the cystlike space is limited at the lung periphery by the visceral pleura. If the visceral pleura be opened over a bulla, the air space beneath will not be lined by squamous cells as is found in a balloon cyst of the congenital type, but a disintegrating lung parenchyma will be seen to make up the walls and base of the bulla. Fine blood vessels completely stripped of supporting lung parenchyma can be seen to cross the air sac. Strands of connective tissue and fine bronchioles also denuded criss-cross the bulla. Multiple small communications with the adjacent bronchi are apparent. Bullae are almost always multiple, but they may be confined to a segment or a lobe. The upper lobes in our experience have been most frequently involved.

Subpleural blebs are the result of rupture of the elastic fibers which bind the pleura to the underlying lung parenchyma.[27] They characteristically occur at the apices of the lung. Small blebs may coalesce to form larger ones, or they may be multiple and discrete and be scattered diffusely over the upper surface of the lung. According to Miller,[27] all cases of subpleural blebs are associated with well-established pulmonary emphysema. Miller also pointed out the similarity between the bleb and the bulla and suggested that the manner of formation is the same. Is it possible that the seemingly innocuous bleb is the precursor of the emphysematous bulla?

Rupture of subpleural blebs is the most frequent cause of spontaneous pneumothorax and is the most common cause for these patients to seek medical attention.[28, 29] In some instances chronic pneumothorax and fibrothorax have resulted from rupture of subpleural blebs and have necessitated decortication to restore pulmonary function.

The ball valve mechanism has been given considerable credit for the progressive enlargement of blebs, bullae, and balloon cysts. Hayashi[30] was able to find microscopic evidence of the valve mechanism which presumably explained the progressive enlargement of air cysts. It is true that a tension cyst or an emphysematous bulla enlarges at the expense of normal lung parenchyma, and the end result is the same as if a perfectly functioning valve were present at the site of the bronchial communication. However, the aerodynamics involved in enlarging bullae and cysts can perhaps be explained in another way. A cyst enlarges because the intracystic pressure builds up to such a degree that the adjacent pulmonary tissue unsupported by a pressure equal to that within the cyst gives way before the enlarging air sac. Intracystic pressure of sufficient force to cause enlargement can result from coughing or straining. Just prior to expulsion of air as in coughing, the glottis is closed and considerable endobronchial pressure is built up by the action of the accessory muscles of respiration. At this time the pressures are equalized throughout all the lung structures that are in communication with an airway, i.e., cyst, bulla, or bleb. When the glottis is opened the endobronchial pressure immediately falls to near zero;[31] however, the relatively small bronchial openings which communicate with the cysts do not allow for an immediate readjustment of the pressure within the cysts and the thin, flaccid walls of the cyst expand at the expense of the adjacent pulmonary tissue. As this process is repeated, the cystic areas enlarge. The thick walls of the bronchogenic type cysts do not allow such ease of expansion. For this reason tension cysts are as a rule not of the bronchogenic type although such a case was recently reported by Allbritten.[12]

Head and Avery[32] described the actions of bullae into which a thoracoscope had been passed. During respiration the bullae could be seen to behave in a paradoxical manner. At the beginning of inspiration the cavity was filled with air whereas expiration caused the cyst walls to bulge inward. These observations would seem to be in conflict with our statements, but it must be pointed out that their observations were made during quiet respiration. Although the effect of a ball valve is present, we have not been able to demonstrate in our series a competent valve mechanism between cyst and bronchus. At operation manual compression of the cyst has in every instance demonstrated the ease with which this type cyst could be collapsed. If our observations are correct it seems improbable that enough pressure can be built up within a cyst by quiet respiration to destroy adjacent lung tissue. To express the problem in another manner, the pressure gradient between the air in the cyst and that in the normally functioning pulmonary tissue must be such that sufficient force is exerted against the cyst wall to cause rupture of the alveolar septa and even the interlobular

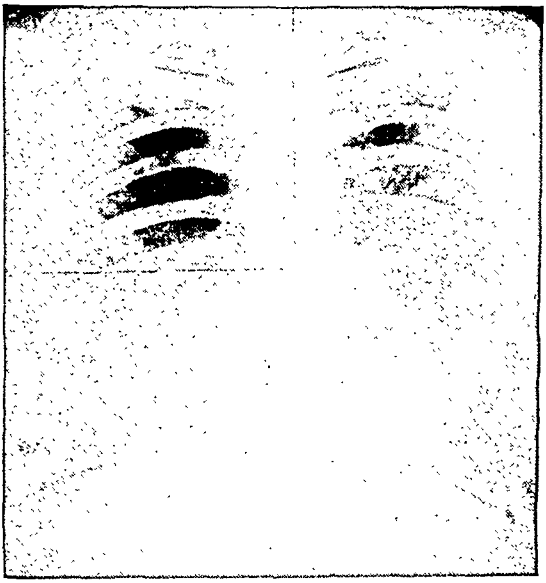

Fig. 17.—(Case 7.) Posteroanterior roentgenogram of the chest. Inset shows lacy marking in the right upper lung field characteristic of bullous emphysema.

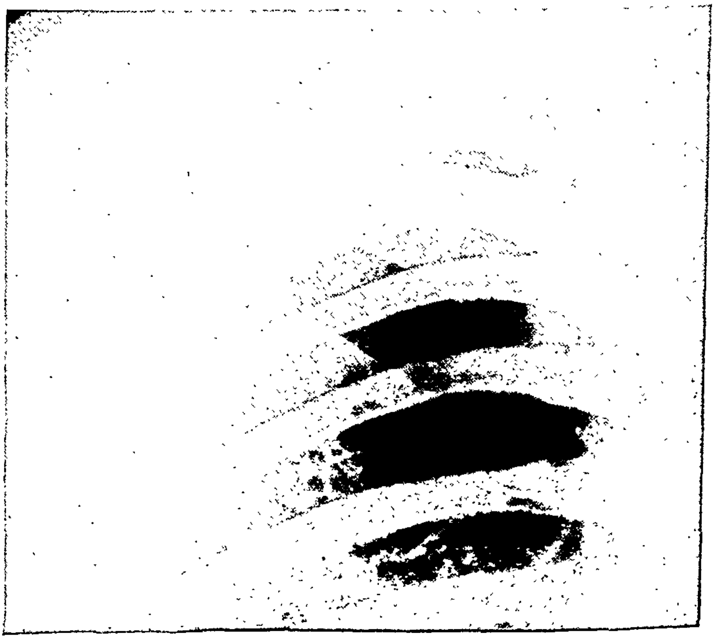

Fig. 18.—(Case 7.) A close-up picture of the inset.

septa. Such pressure can easily be built up in the pulmonary system by coughing, but at the time the glottis is closed the pressure is equal throughout the entire thoracic cavity and it is only upon sudden release of this pressure by opening the glottis that the pressure gradient between the air within the cyst and the air in the normal lung parenchyma becomes sufficient to allow expansion of the cyst.

The origin of pulmonary bullae has been thought to be due to inflammation and partial occlusion of the smaller air passages. This must undoubtedly play a large part. In all of the cases of bullous emphysema in our series a severe, chronic cough has been present.

The surgical treatment of selected cases of pulmonary blebs and emphysematous bullae has in recent years become well established.[3, 12, 32-36] The following cases are representative of the group that have been treated surgically. The results of this treatment have been uniformly good and all patients have been returned to military duty with one exception; the case history of that patient is reported below.

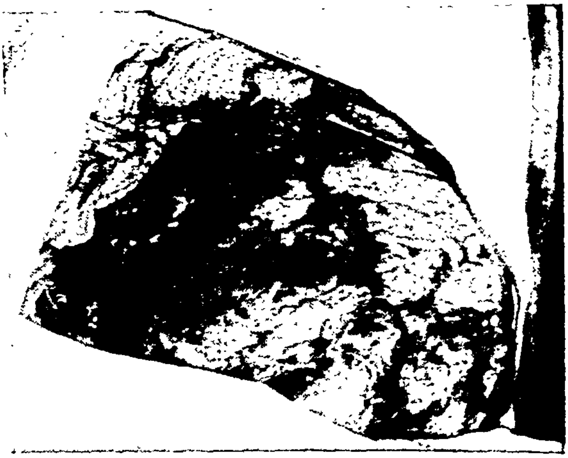

Fig. 19.—(Case 7.) The right upper lobe at operation has almost been completely replaced with large emphysematous bullae.

CASE 7.—A 34-year-old white man complained of a cough which was productive in the morning of a small amount of sputum and slight dyspnea on exertion. In the morning his cough was so severe that he frequently vomited. The exertional dyspnea was particularly noticeable because the patient had been an instructor in a Ranger Battalion during the war and at which time he had undergone rigorous training with no apparent difficulty. He volunteered the information that in recent years he smoked three packages of cigarettes per day. Chest roentgenograms revealed emphysema, bullous type, involving both upper lobes, more marked on the right (Figs. 17 and 18). Right thoracotomy was performed, and the right upper lobe was found to be almost completely replaced by emphysematous bullae (Fig. 19). The right middle and right lower lobes were free of disease. The multiple cysts had enlarged to such a degree after the pleural adhesions were lysed that the pulmonary hilum could not be adequately exposed until the cysts were collapsed by manual compression and clamps placed across the base of the cysts. The bullae were easily compressed, and no valve mechanism was apparent. Lobectomy was completed without incident.

Forty-eight hours postoperatively the patient developed atelectasis on the right which necessitated aspiration bronchoscopy. Despite repeated aspiration of the airway, his mediastinum failed to shift back to its normal position. The heart was markedly displaced, and the patient developed paroxysmal auricular tachycardia. Frequent bronchoscopic aspirations of the right main-stem bronchus failed to relieve the marked mediastinal shift. It was assumed that the bullae in the left apex had become overdistended at the time of the onset of atelectasis and that, despite all efforts to reduce the size of these bullae, they continued to impinge on normal lung and mediastinal structures to such a degree that the existence of the patient was threatened. In a desperate effort to reduce the bullae in the left apex, a trochar was placed through the left anterior chest and a catheter was placed in the largest presenting bulla. Suction was applied (Fig. 20).

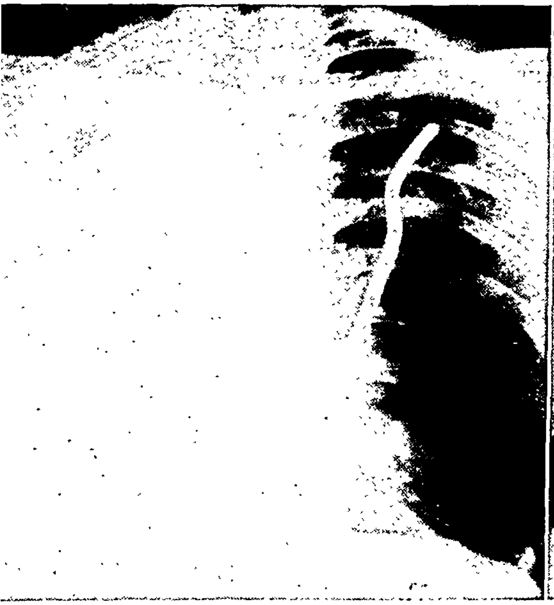

Fig. 20.

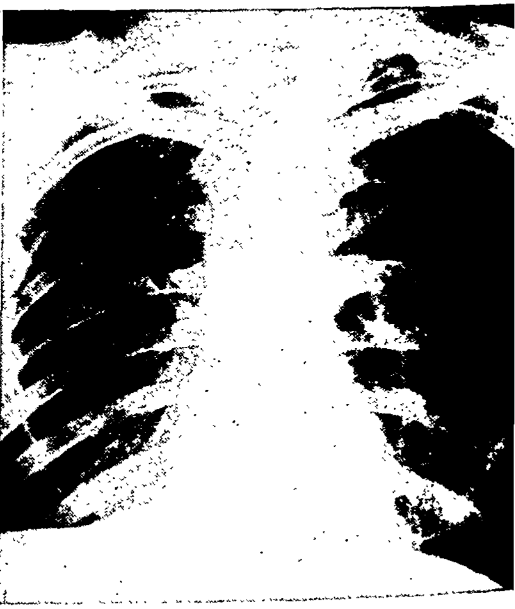

Fig. 21.

Fig. 20.—(Case 7.) Portable roentgenogram of the chest showing intercostal catheter into a large bulla in the left apex. The marked mediastinal shift is evidenced.

Fig. 21.—(Case 7.) Postoperative posteroanterior roentgenogram of the chest showing good expansion of the remaining pulmonary segments. At this time the patient regained vital capacity of 4.5 liters.

This failed to relieve the situation, and on the fifth postoperative day, three days after the onset of these most distressing complications, a thoracotomy was performed on the left and numerous large bullae involving the anterior and apical posterior segments of the left upper lobe were found. There was no involvement of the lower lobe. As soon as the chest was opened, the patient under endotracheal anesthesia improved considerably. The bullae were resected. Despite the occurrence of pneumothorax on the left side which required that suction be applied to the thoracotomy tubes, in order that adequate ventilation of the lungs could take place, the patient recovered.

*Pathologic diagnosis:* Bullous emphysema, bilateral, confined to the upper lobes.

*Comment.*—This patient represents a case of bullous emphysema localized to the upper lobes of both lungs. It is interesting to note that he apparently was free of disease five years prior to his admission to the hospital, at which time he had demonstrated repeatedly his ability to do the most strenuous type of physical exertion. At that time it must be presumed that he had no pulmonary disease. It is also interesting to speculate as to what role violent cough-

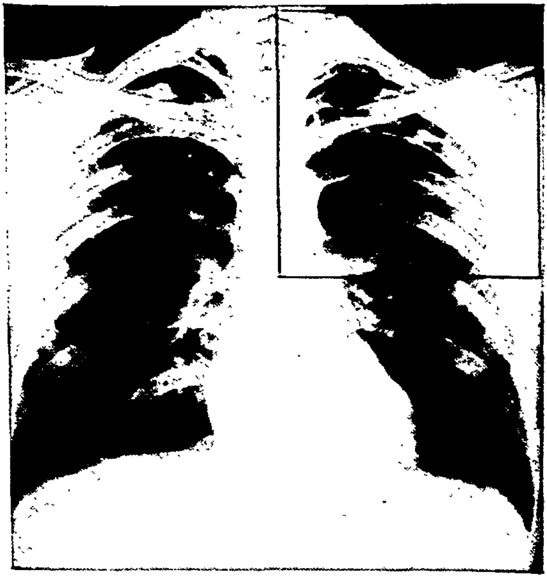

Fig. 22.—(Case 8.) Posteroanterior roentgenogram of the chest. Inset shows characteristic appearance of bullous emphysema.

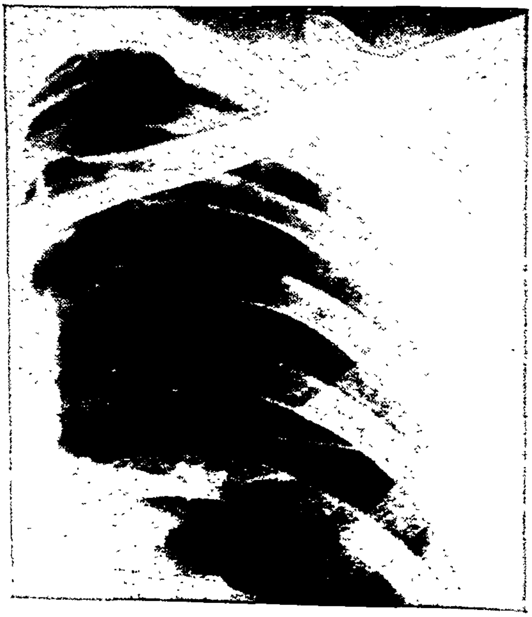

Fig. 23.—(Case 8.) Close-up of inset.

ing played in the production of the tremendous emphysematous bullae found at operation. The rather serious complications which arose as a result of his having bilateral bullae necessitated a thoracotomy on the left five days after a right upper lobectomy. Such a complication reported by Dugan and Samson[28] caused a fatality in their series. They suggested that perhaps a catheter placed into the expanding bulla would relieve the tension and prevent the compression of vital structures. This was tried in our patient and failed, probably due to the large number of bullae which were present and as one collapsed others expanded to fill the space provided. Although this patient did not return to duty because of the uncertain prognosis of his disease, he did regain a vital capacity of 4.5 liters and is living a useful and active life (Fig. 21).

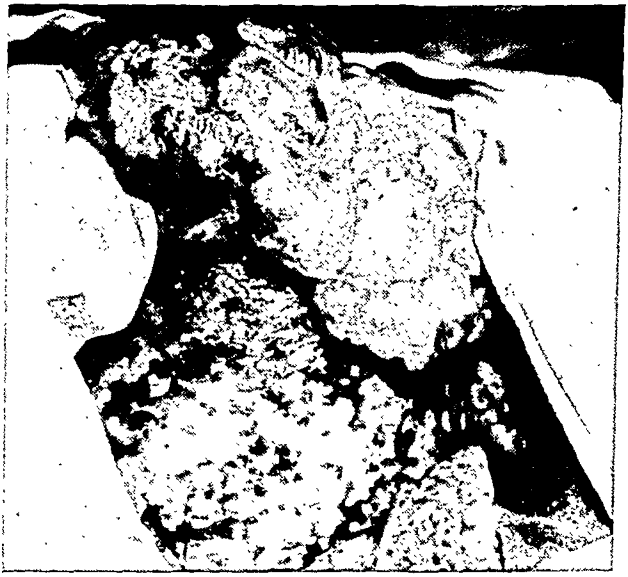

Fig. 24.—(Case 8.) At operation large emphysematous bullae replaced the apical posterior segment.

Case 8.—This 39-year-old white man had a chronic, slightly productive cough for ten years which he attributed to smoking cigarettes. Routine x-ray examination had revealed emphysema localized to the left upper lobe (Figs. 22 and 23). Increase in the size of the bullae was noticed when the films were compared to roentgenograms taken on previous admission. Bronchography outlined a normal tracheobronchial tree. Because of the localized nature of the lesion, exploratory thoracotomy was thought to be indicated. At operation, large emphysematous bullae had almost completely replaced the apical posterior segment of the left upper lobe (Fig. 24). There was no other evidence of disease in the chest. The bullae were resected. The patient was returned to duty.

*Pathologic diagnosis:* Emphysema, bullous type, with alveolar bleb formation.

*Comment.*—This case represents the type of bullous emphysema which is localized to a segment and which lends itself well to surgical removal. This is classified, of course, as acquired type of pulmonary cysts.

The following two cases were classified as emphysematous blebs:

CASE 9.—A 28-year-old white soldier was admitted to the hospital after his third episode of spontaneous pneumothorax. He had had repeated surveys for tuberculosis, all of which had been negative. There was no evidence of disease other than the pneumothorax which was the transfer diagnosis. On examination of the roentgenograms, a small bleb could be seen at the left apex (Fig. 25). A thoracotomy was performed, the bleb was excised, and the surface of the upper lobe was powdered with sterile talc. The patient made an uneventful recovery and was returned to duty.

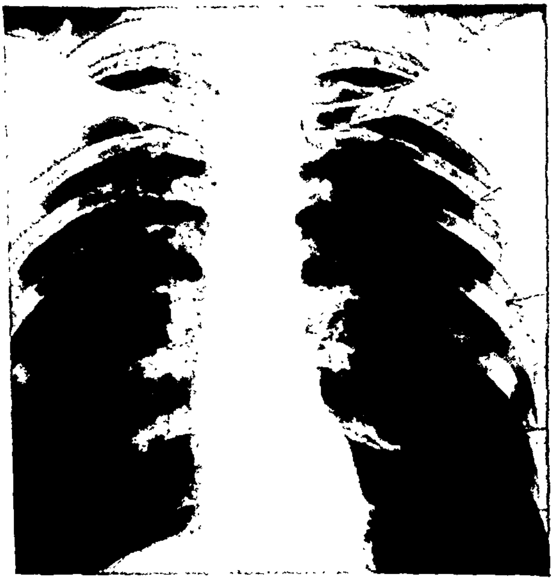

Fig. 25.—(Case 9.) Posteroanterior roentgenogram of the chest showing pneumothorax on the left and at the apex an emphysematous bleb.

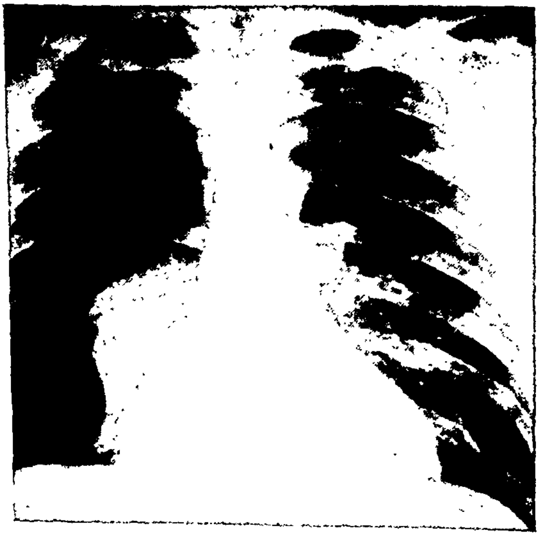

Fig. 26.—(Case 10.) Posteroanterior roentgenogram of the chest reveals pneumothorax on the right, and very faintly seen a large emphysematous bleb.

CASE 10.—A 29-year-old white man was admitted to the hospital following a spontaneous pneumothorax (Fig. 26). On chest x-ray examination, a large bleb could be seen at the uppermost portion of the collapsed right lung. Tuberculosis survey was negative. There was no evidence of disease. At operation a large subpleural bleb (Fig. 27) was excised, and the surface of the upper lobe was poudraged. The patient made an uneventful convalescence and was discharged to duty.

*Comment.*—The two preceding cases are representative of the type of subpleural blebs which are the most frequent cause of spontaneous pneumothorax. It has been our policy when confronted with the problem of spontaneous pneumothorax in otherwise healthy individuals to treat the pneumothorax conservatively. Many of these cases, of course, have not had recurrence. However, in patients who have recurrent spontaneous pneumothorax on three or more occasions and in those in which the subpleural blebs can be visualized, we have elected to do a thoracotomy with excision of the bleb or blebs and poudrage of the lung surface. The poudrage serves the purpose of causing the parietal and visceral pleura to become adherent in that region and also tends, because of the inflammatory reaction, to cause a thickening of the visceral pleura and lessen the tendency of rupture of a bleb. The patients treated in this manner have all been returned to military duty.

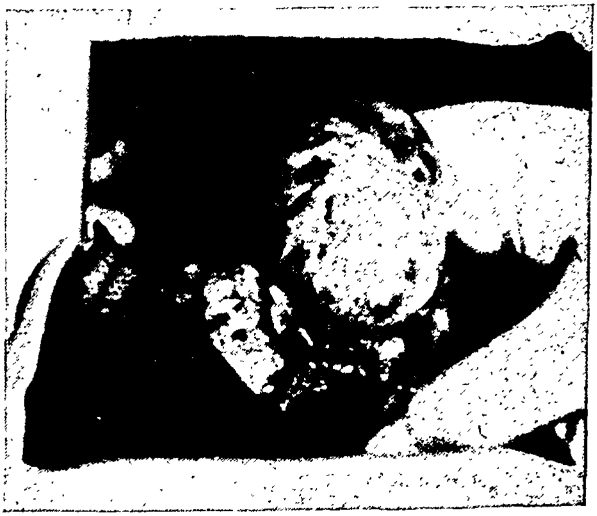

Fig. 27.—(Case 10.) At operation the large solitary bleb is demonstrated.

## SUMMARY

The incidence of cystic lung disease is much greater than is indicated by figures based on routine roentgenogram or necropsy examinations. A classification of the various types of cysts has been proposed. A standardization of the nomenclature would facilitate statistical analyses and help clarify much of the confusion in regard to prognosis and treatment. Some of the many problems occurring in the surgical management of these cases have been discussed.

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# THE FATE OF AN ISOLATED SEGMENT OF THE ESOPHAGUS

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WINSTON-SALEM, N. C.

RESECTION of the esophagus is very difficult when there is marked fixation of the esophagus to the adjacent tissue. Such fixation is especially severe in cases of caustic stricture, and in carcinoma if the growth has spread beyond the esophageal wall. The advisability of leaving the involved segment of the esophagus in place in such cases and construction of a side-tracking anastomosis, either esophagoesophageal or esophagogastric, was considered, and an experiment set up to evaluate this procedure in dogs.

## METHOD

Nine mongrel dogs were used. In order to provoke adhesions to the segment of esophagus that was to be isolated, and so to simulate actual conditions, right thoracotomy was performed, and a 5 cm. segment of the lower thoracic esophagus wrapped with a single layer of cellophane which had been previously tested for tissue irritability (active agent dicetyl phosphate). Approximately four weeks later, when esophagoscopy demonstrated a narrowing of the lumen of the esophagus at the site of the wrapping, left thoracotomy was performed. In each animal there were dense adhesions to the esophagus at the level of the cellophane wrapping. The esophagus was divided 1 cm. above and 1 cm. below the involved area, and an anastomosis was constructed between the proximal esophagus and the distal esophagus or stomach (in three cases the stricture was so low as to necessitate the use of the cardia for the anastomosis). In every case the diaphram was opened and a sufficient amount of stomach brought into the chest to allow the anastomosis to be constructed without undue tension. In Group I dogs the isolated segment of the esophagus was closed at each end with continuous silk sutures. In Group II dogs each end of the isolated segment was left open. In Group III dogs the segment was split lengthwise so as to leave an ''opened tube'' of esophagus. The bleeding vessels at the cut edges were clamped and tied. The operations were performed with aseptic technique. Each dog received subcutaneous fluids the day of operation, water and milk for the first to the seventh postoperative day, and regular dried kennel rations thereafter.

## RESULTS

At varying intervals (Table I) after the second operation the dogs were sacrificed for further study. In every case, save that of dog 253 of Group III, the isolated segment of the esophagus had closed off to become a mucus-filled cyst (Fig. 1). The cysts varied in size from 3 to 6 cm. in diameter (Table I), with an average size of 4.5 cm. The material in the cyst in every instance was a thin, mucoidal, yellow colored, odorless fluid. The esophagoesophageal or esophagogastric junction was in good condition in each case. Microscopic sections of the walls of the cysts showed an extremely thin layer of squamous epithelium backed by a layer of connective tissue and muscular tissue; there were no recognizable glandular elements (Fig. 2).

The formation of the cysts in Groups II and III dogs can be explained by adherence of the raw edges which came into contact. Dog 253, explored at

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*Trainee, National Cancer Institute.

TABLE I

| TREATMENT OF ISOLATED SEGMENT | DOG NO. | POSTOPERATIVE DAYS AFTER ISOLATION OF SEGMENT | SIZE OF CYST (CM.) |
|---|---|---|---|
| Group I<br>(Both ends of<br>segment closed) | 250<br>256<br>260 | 12<br>98<br>221 | 4<br>5<br>4 |
| Group II<br>(Both ends of<br>segment open) | 254<br>255<br>259 | 72<br>128<br>240 | 6<br>6<br>3 |
| Group III<br>(Segment split<br>lengthwise) | 253<br>258<br>261 | 14<br>110<br>219 | no cyst<br>4<br>4 |

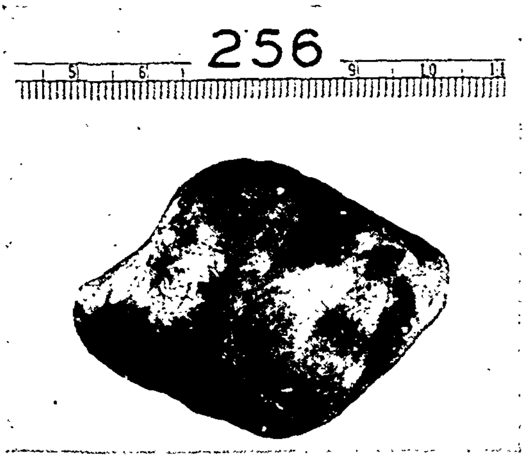

Fig. 1.—*Photograph of esophageal cyst removed from Dog No. 256, ninety-eight days postoperatively.*

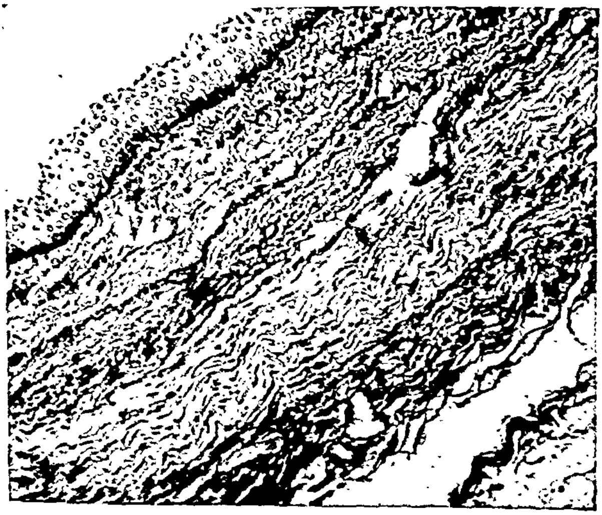

Fig. 2.—Photomicrograph of a section of the wall of the cyst shown in *Fig. 1*. The stratified epithelium, submucosa, and muscularis are plainly delineated, but there is a complete lack of glandular tissue. (Hematoxylin and eosin, ×95.)

# THE FATE OF AN ISOLATED SEGMENT OF THE ESOPHAGUS

W. RALPH DEATON, JR., M.D.,* AND H. H. BRADSHAW, M.D.
WINSTON-SALEM, N. C.

RESECTION of the esophagus is very difficult when there is marked fixation of the esophagus to the adjacent tissue. Such fixation is especially severe in cases of caustic stricture, and in carcinoma if the growth has spread beyond the esophageal wall. The advisability of leaving the involved segment of the esophagus in place in such cases and construction of a side-tracking anastomosis, either esophagoesophageal or esophagogastric, was considered, and an experiment set up to evaluate this procedure in dogs.

## METHOD

Nine mongrel dogs were used. In order to provoke adhesions to the segment of esophagus that was to be isolated, and so to simulate actual conditions, right thoracotomy was performed, and a 5 cm. segment of the lower thoracic esophagus wrapped with a single layer of cellophane which had been previously tested for tissue irritability (active agent dicetyl phosphate). Approximately four weeks later, when esophagoscopy demonstrated a narrowing of the lumen of the esophagus at the site of the wrapping, left thoracotomy was performed. In each animal there were dense adhesions to the esophagus at the level of the cellophane wrapping. The esophagus was divided 1 cm. above and 1 cm. below the involved area, and an anastomosis was constructed between the proximal esophagus and the distal esophagus or stomach (in three cases the stricture was so low as to necessitate the use of the cardia for the anastomosis). In every case the diaphram was opened and a sufficient amount of stomach brought into the chest to allow the anastomosis to be constructed without undue tension. In Group I dogs the isolated segment of the esophagus was closed at each end with continuous silk sutures. In Group II dogs each end of the isolated segment was left open. In Group III dogs the segment was split lengthwise so as to leave an ''opened tube'' of esophagus. The bleeding vessels at the cut edges were clamped and tied. The operations were performed with aseptic technique. Each dog received subcutaneous fluids the day of operation, water and milk for the first to the seventh postoperative day, and regular dried kennel rations thereafter.

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a dog 221 days postoperatively (dog 200). Thus the supposition that the isolated segment might become of tremendous size and so adversely affect the respiratory and circulatory functions is apparently baseless. Further, in patients who have advanced carcinoma of the esophagus in whom a palliative side-tracking procedure is planned, a mediastinal cyst is of no consequence.

## CLINICAL APPLICATION

Since completion of the above work, one patient has presented himself for treatment of far-advanced carcinoma of the esophagus. The protocol is as follows:

J. W. A., a 65-year-old farmer, was admitted to the North Carolina Baptist Hospital on May 23, 1951. At that time he gave a history of having regurgitated some watermelon eight months previously. Soon after that, solids began to be intermittently regurgitated, and for the month before admission he had been unable to eat any solid food. Although his appetite had remained excellent, he had lost about 60 pounds in weight since the onset of the illness. In April a roentgenographic examination of the esophagus was performed, and revealed a filling defect with almost complete obstruction in the midesophageal region (Fig. 3). He was subsequently referred to the North Carolina Baptist Hospital for therapy. Physical examination showed an elderly gaunt man who was quite pleasant and cooperative. Blood pressure was 130/80. There was evidence of considerable weight loss, but no other positive findings. Accessory clinical findings demonstrated no abnormality. Esophagoscopy was performed, and at 30 cm. from the upper incisor teeth was found a fungating easily bleeding mass which completely encircled the esophagus. Biopsy showed anaplastic carcinoma (Fig. 4). The following day left thoracotomy was performed. A hard, irregular growth was found to circumscribe the esophagus at the arch of the aorta. The growth was firmly fixed to the aorta, trachea, and prevertebral fascia. Enlarged lymph nodes were present above and below the tumor. It was impossible to free up the esophagus without risking an exsanguinating hemorrhage, and, as a cure was impossible, it was decided to leave the growth in place and construct an esophagogastric anastomosis around it. This was easily

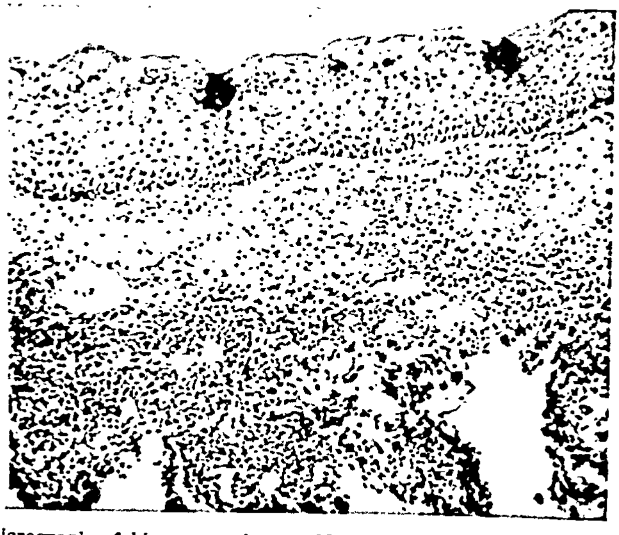

Fig. 4.—Photomicrograph of biopsy specimen. Note the intact epithelium, beneath which the submucosa and muscularis are infiltrated with carcinoma. (Hematoxylin and eosin, ×95.)

fourteen days, had not had time to seal off the isolated segment. However, the segment had rolled on itself so as to form a tube. Undoubtedly the opposing edges would have become adherent within a matter of days.

## DISCUSSION

From abroad physicians have reported the construction of side-tracking esophagogastric anastomoses, for circumventing strictured areas of the esophagus, in cases of lye strictures and carcinoma.[1-3] However, all cases reported have had the esophagus left intact, with performance of an intrathoracic side-to-side esophagogastrostomy. The leaving of the esophagus attached to the stomach makes it necessary to free up the stomach to a much more extensive degree than simple isolation of the involved segment of the esophagus, but the authors apparently believe that the continuity is desirable for drainage. If cyst formation is to be averted, evidently drainage must be provided for. However, the fact that the animals in this experiment lived and did well, with no apparent injurious effects from the mediastinal cysts, indicates that the cyst formation may not be detrimental. Apparently the size of the cyst is self limited, for as the pressure in the cyst increases, the secreting glands atrophy and the size of the cyst becomes stationary. Otherwise, a cyst removed from a dog twelve days postoperatively (dog 250) would not be the same size as a cyst removed from

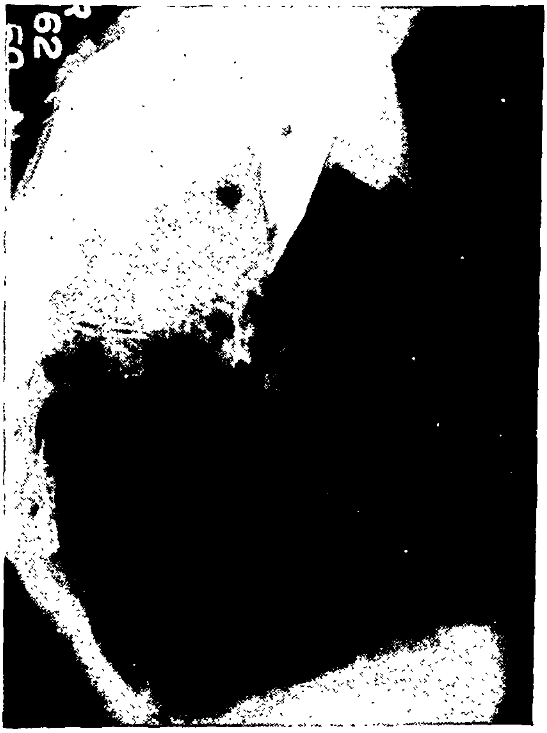

Fig. 3.—Lateral chest roentgenogram following barium swallow. The esophagus is almost completely obstructed, and there is some overflow of the barium into the trachea.

a dog 221 days postoperatively (dog 200). Thus the supposition that the isolated segment might become of tremendous size and so adversely affect the respiratory and circulatory functions is apparently baseless. Further, in patients who have advanced carcinoma of the esophagus in whom a palliative side-tracking procedure is planned, a mediastinal cyst is of no consequence.

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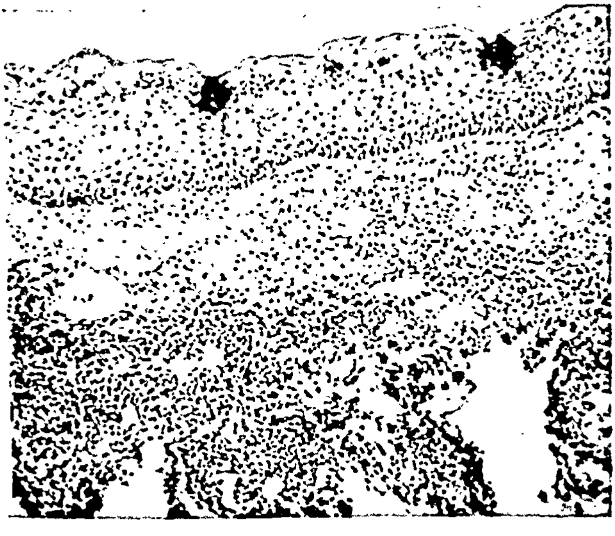
Fig. 4.—Photomicrograph of biopsy specimen. Note the intact epithelium, beneath which the submucosa and muscularis are infiltrated with carcinoma. (Hematoxylin and eosin, ×95.)

done without undue incident. Postoperatively he did quite well, being able to take water by mouth on the first postoperative day, a full liquid diet on the third postoperative day, and a soft diet on the fifth day. Fluoroscopy on the tenth day showed the esophagogastric anastomosis to be functioning well, with no widening of the mediastinum indicative of fluid accumulation in the isolated segment of the esophagus. He was discharged on the twelfth postoperative day. At home he continued to enjoy eating, and gained weight.

## CONCLUSIONS

In dogs it has been shown that isolation of a strictured segment of the esophagus produces a cyst, regardless of the treatment accorded the segment. The size of the cyst is apparently self limited, being regulated by the amount of pressure in the cyst, as the pressure causes atrophy of the secreting glands. The presence of a small mediastinal cyst did not adversely affect any of the dogs. Clinically, it is believed that all cases of carcinoma of the esophagus, unless distant metastases are demonstrated, should be explored. If the lesion is found to be too extensive for a curative procedure, and unfortunately a great number are, then a palliative esophagoesophageal or esophagogastric anastomosis appears to be indicated. This is a relatively short procedure, and will allow the patient to eat normally for the remainder of his life. Death from the complications of extension of the cancer is easier on the patient and his family than starvation. Gastrostomy gives no satisfaction to the patient, and is difficult to attend to.

## SUMMARY

Isolation of a segment of the esophagus in the dog results in a small asymptomatic esophageal cyst. Such a procedure was performed on one patient, with quite satisfactory results. However, post-mortem studies on long-term survivors will be necessary before one can conclude that the operation is as satisfactory in human beings as in dogs.

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# PULMONARY TUBERCULOSIS FOLLOWING RESECTION FOR NONTUBERCULOUS DISEASE

EMIL ROTHSTEIN, M.D.,* AND CHARLES E. GERSON, M.D.**
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IN THE past two decades, during which excisional surgery for pulmonary lesions has been performed with increasing frequency, there have been many articles dealing with techniques, indications, and results. Immediate and early complications arising after resection have been stressed. One late complication or sequel has received very little emphasis in medical literature. This is the delayed development of pulmonary tuberculosis. The occurrence of two such cases in our experience and the rapidly progressive nature of the disease in both patients aroused our interest in this problem. In addition, the coincidental finding in several patients of pulmonary tuberculosis and nontuberculous bronchiectasis led us to re-evaluate our therapeutic indications when these two disease processes both are in need of treatment.

CASE 1.—L. A. C., aged 30 years, had several attacks of hemoptysis and pneumonia in 1941, 1945, and 1947. Bronchograms (Fig. 1) showed definite bronchiectasis involving the superior segment of the right lower lobe. Right lower lobectomy was performed uneventfully in September, 1947. Sputum examinations before surgery failed to reveal tubercle bacilli, nor were tuberculous changes present in the resected specimen. Subsequent roentgenograms in 1947 showed no evidence of pulmonary tuberculosis. A roentgenogram in November, 1948, showed infiltration in the remainder of the right lung, which rapidly progressed to cavitation and extensive consolidation (Fig. 2, April 27, 1949). Sputum specimens were repeatedly positive for tubercle bacilli. Despite five months of streptomycin therapy, the disease remained largely unchanged. On Feb. 3, 1950, the rest of the right lung was removed. Far-advanced tuberculosis was found in the resected lung. After a protracted course characterized by empyema, tuberculous pericarditis, and contralateral pleural effusion, the patient expired on Aug. 24, 1951.

CASE 2.—B. M., 25 years of age, had left lower lobe bronchiectasis since 1943 (Fig. 3, Jan. 16, 1946). Left lower lobectomy was done in April, 1947. Sputum examinations before surgery failed to reveal tubercle bacilli; no tuberculous changes were found in the resected specimen. Postoperative films revealed no evidence of pulmonary tuberculosis. A subsequent roentgenogram in April, 1948 (Fig. 4), showed far-advanced tuberculosis in the left upper lobe. The patient was treated with streptomycin, and a left thoracoplasty was performed in August, 1948. He is still seriously ill with active disease in the other lung.

## DISCUSSION

The only article we could find dealing directly with this subject was by Katz.[1] He reported four cases in which resections were done for bronchiectasis, sarcoid granuloma, and actinomycosis. In each of these cases sputum studies

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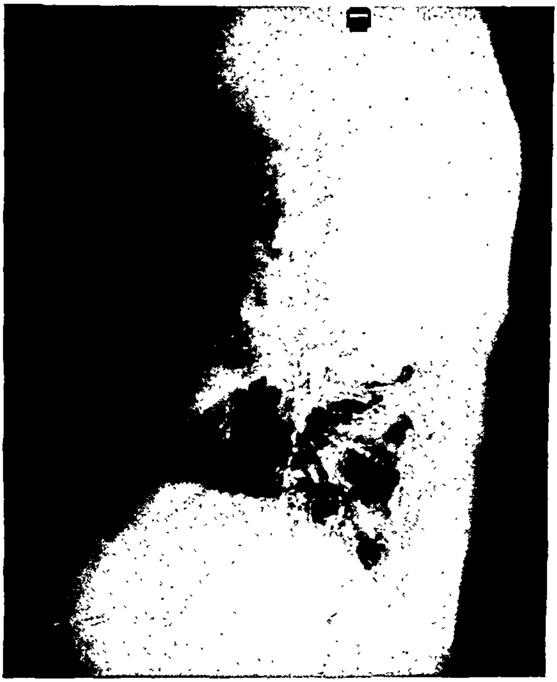

Fig. 1.—(Case 1, Sept. 26, 1947.) Bronchogram revealing saccular bronchiectasis in the dorsal division of the right lower lobe bronchus.

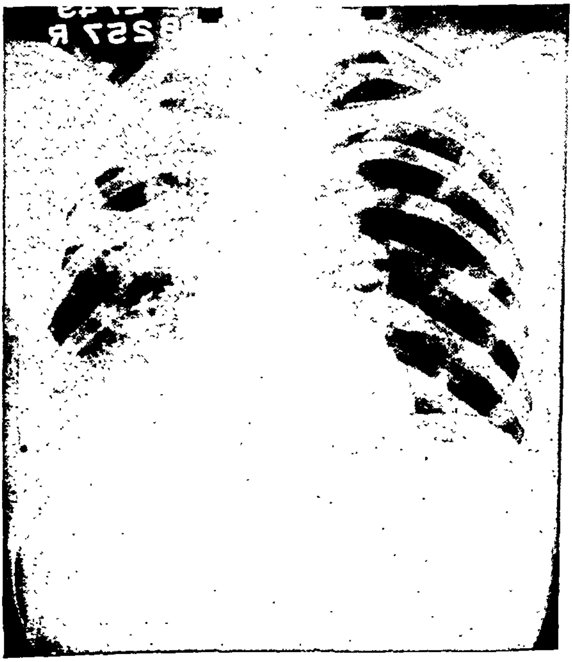

Fig. 2.—(Case 1, April 27, 1949.) Far-advanced pulmonary tuberculosis, involving right upper and middle lobes.

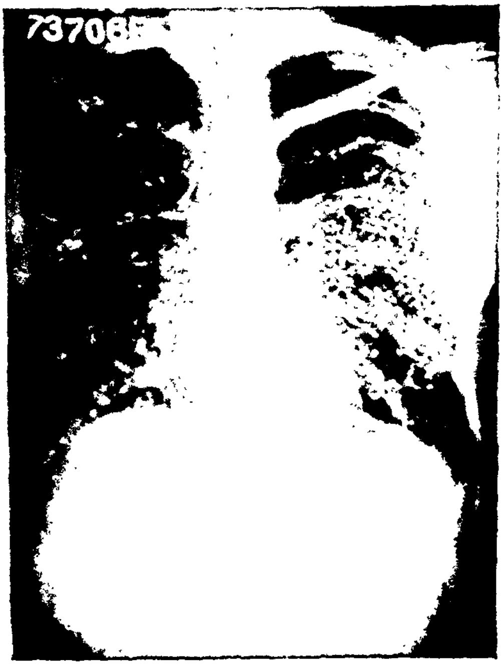

Fig. 3.—(Case 2, July 16, 1946.) Bronchogram revealing extensive saccular bronchiectasis in the basal divisions of the left lower lobe.

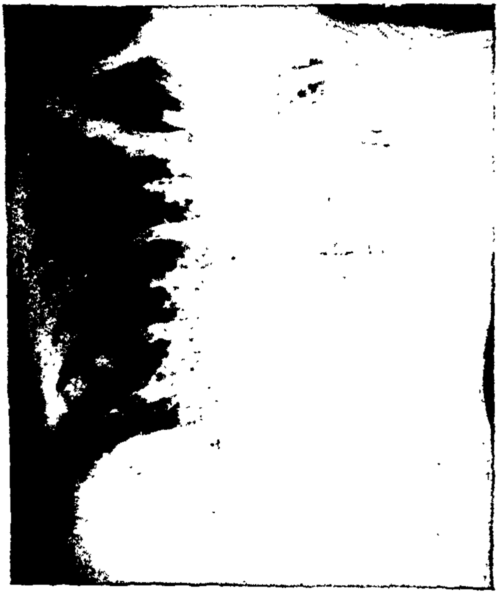

Fig. 4.—(Case 2, April 21, 1948.) Far-advanced pulmonary tuberculosis in the left upper lobe.

prior to resection were negative for tubercle bacilli, and the resected specimens showed no evidence of tuberculosis. Postoperative roentgenograms failed to reveal pulmonary tuberculosis. In the three cases having had lobectomy performed, far-advanced caseocavitary disease appeared in the remaining lobe in less than one year after resection. Our two patients presented exactly the same picture.

Other reports dealing with this subject have been buried in statistics pertaining to resections for bronchiectasis. Thus Maier[2] reported on sixty-four cases treated by resection. Active pulmonary tuberculosis developed in two patients. One of these had had inactive tuberculosis before surgery, and the other had shown no previous evidence of pulmonary tuberculosis. Sellors, Thompson, and Qvist[3] reported 100 resections for bronchiectasis. Seven of these patients developed pulmonary tuberculosis. Four of the resected specimens revealed evidence of tuberculous changes in addition to the bronchiectasis. Three of the seven patients had died of tuberculosis at the time of their report. Lambert[4] reported seventy-eight cases, of which ten developed pulmonary tuberculosis. The serious nature of this clinically evident tuberculous disease was shown in his series by the fact that four required thoracoplasty, two have died, and of the eight remaining alive, five still had positive sputa at an unspecified interval after resection. Six of these ten cases developed within one year after resection and the other four from sixteen to thirty months postoperatively. In four of the postlobectomy cases the disease developed in the remaining portion of the homolateral lung, as in Katz' series and in our cases.

Despite this very appreciable incidence, the subject has apparently aroused but little interest. This was shown in part by the fact that none of the discussants of Lambert's paper[4] commented upon the high incidence of this very serious complication.

The exact pathogenesis of the tuberculous lesion is of course impossible to state. The reason for any given patient developing pulmonary tuberculosis is such a compound of bacteriologic, immunologic, and mechanical factors that in the small groups studied the possibility of coincidence cannot be ruled out. Certain features, however, suggest that this is not the case in the subject under discussion.

In almost every reported case, review of the preoperative films showed no evidence of pulmonary tuberculosis. None of the authors refers to his gross findings at the time of surgery, and there is nothing to suggest that there were gross tuberculous changes present in the unremoved lung tissue remaining after lobectomy. Early postoperative films similarly failed to reveal shadows typically associated with pulmonary tuberculosis. Sputum examinations, whenever reported, were negative for tubercle bacilli, and in most instances pathologic examination of resected specimens showed no evidence of tuberculosis.

In our two cases the tuberculosis appeared on the side which had been subjected to resection, and did so within one year of surgery. The disease rapidly became far advanced and did not respond to medical measures. Katz' three postlobectomy cases followed exactly the same pattern, as apparently did four of Lambert's patients.

Several possibilities for the development of tuberculosis in this group must be considered. Coincidence has been mentioned but should probably be rejected. Lowered resistance due to chronic infection or due to the surgery must be mentioned as a possibility and can in no case be completely discounted. Undetected tuberculosis may have been present in the lobe removed and the disease may have been spread to the remaining lung parenchyma at operation. That this is unlikely seems evident from the fact that postoperative examination of resected specimens failed, in most instances, to reveal tuberculous changes. If sufficient caseation and cavitation were present to produce such a spread, they could hardly escape detection by the pathologist. Other circumstances mitigating against this hypothesis are the negative sputum examinations for tubercle bacilli before and after surgery and the delayed (six to twelve months) appearance of the tuberculous lesions. Postoperative spreads following surgery for pulmonary tuberculosis usually are noted in the immediate or early postoperative period.

Tuberculous disease may have been present in the hilar structures, and if such tissue were cut across at surgery, this might explain the late development of tuberculosis in the adjacent lung. This hypothesis was advanced by Sellors, Thompson, and Qvist,[3] and is discussed by Lambert.[4] The use of individual ligation at the bronchial stump will presumably make unlikely section of grossly involved tuberculous nodes or other tuberculous tissue without immediate discovery by the surgeon.

Distention of remaining pulmonary tissue, containing hidden unhealed tuberculous foci must be very strongly indicted as the most probable cause for the phenomenon under discussion. We were unable to find any experimental proof that distention of such lung tissue might actually result in progression of a latent tuberculous process. There are, however, clinical grounds for such an assumption. One of these is the beneficial effect upon an active tuberculous process of a decrease in pulmonary volume and tension, as provided by pneumothorax, phrenic nerve surgery, pneumoperitoneum, and thoracoplasty. While these methods of treatment may exert their therapeutic effects in various ways, one of the most important of these is by the opposite of overdistention, i.e., by relaxation of the involved pulmonary tissue. It is therefore reasonable to assume that overdistention may, in some cases, delay healing and predispose to progression of disease. This factor is recognized in the generally accepted practice of performing thoracoplasty after upper lobectomy or pneumonectomy for pulmonary tuberculosis, to avoid overdistention of the remaining lung tissue.

We have allowed the clinical material and theoretical considerations presented above to assist us in formulating certain tentative indications for the treatment of patients who present roentgenographic evidence of pulmonary tuberculosis in addition to some other disease requiring excisional surgery of a lobe or lung. These indications are based upon two features. One of these is the need to consider the tuberculous condition as potentially serious, if it is present in lung tissue which will be subjected to postresection distention. The other is based upon the work of Baum and Baum.[5] These authors showed that the coincidental presence of a suppurative pneumonia and tuberculosis often resulted in a harmful effect upon the tuberculous process. They found that when a suppurative pneumonia (such as is commonly associated with chronic lung abscess, bronchiectasis, bronchostenosis, etc.) was present in the same lobe as the pulmonary tuberculosis, 70 per cent of the tuberculous lesions were unfavorably influenced. When the processes were present in different lobes the incidence of reactivation or spread of the tuberculosis was less, but a suppurative pneumonitis apparently may exert a harmful effect upon pulmonary tuberculosis wherever situated. We consider the Baums' work to favor the principle of excising pulmonary suppurating or obstructive lesions coexisting with pulmonary tuberculosis, when this can be done without causing overdistension of the tuberculous areas.

The indications formulated below are, of course, tentative only, since they are based on an admittedly small amount of clinical material.

1. The principle of conservation of normal lung tissue should always be adhered to. Segmental resection when feasible should always be preferred to lobectomy.

2. When inactive pulmonary tuberculosis is present steps should be taken to avoid overdistention of the tuberculous area. Thus after lower lobe resection, phrenic paralysis possibly associated with a period of pneumoperitoneum would be desirable. If the lesions coexist in the same lobe, this should be considered a further indication for resection.

3. In the presence of active pulmonary tuberculosis, distention of the tuberculous area must be regarded as potentially very dangerous. If the disease processes can be excised at the same operation this should again be considered a positive indication for lobectomy or pneumonectomy. When the disease processes are bilateral we feel that prolonged sanatorium treatment of the tuberculous process with appropriate chemotherapy should be the method of choice.

Since this study was begun, three patients with bilateral lower-lobe bronchiectasis and active upper-lobe tuberculosis have presented themselves for therapy. In each case, in the absence of a tuberculous lesion, segmental resections or lobectomies would have been performed for the bronchiectasis. Guided by the above principles, however, each patient was treated with chemotherapy (consisting of 1 Gm. streptomycin twice weekly and 12 Gm. PAS daily) for prolonged periods of time (eight to eighteen months) with clinical arrest of the tuberculous lesion and marked improvement in the symptoms of bronchiectasis.

## CONCLUSIONS

Pulmonary tuberculosis occurs as a sequel with impressive frequency in patients who have had resection for nontuberculous disease, even in the face of negative roentgenograms and bacteriologic studies for tubercle bacilli in the immediate pre- and postoperative periods. In several series the reported incidence varied from 3 to 13 per cent. This finding leads to the conclusion that this is a group needing periodic follow-up roentgenograms in much the same way as contacts to active cases of tuberculosis require follow-up. Routine chest roentgenograms every three months for one year after surgery and every six months for several years longer might prevent the tragic development of a tuberculous process already far advanced when first detected.

The exact pathogenesis of this tuberculous process is not known, but overdistention of the remaining lung tissue seems at present the most important single factor. Therefore, when pulmonary tuberculosis is known to coexist with some other disease requiring resection, overdistention should, if possible, be avoided. This may be effected by a combination of segmental resection whenever possible, with consequent conservation of lung tissue, and diminution in the size of the hemithorax by some combination of phrenic nerve surgery, pneumoperitoneum, thoracoplasty, etc. Such patients should in addition be followed with frequent roentgenogram examinations in order to discover any reactivation of the tuberculous process as soon as possible after its development.

## SUMMARY

1. Two cases are presented in which far-advanced pulmonary tuberculosis developed in patients who had recently undergone lobectomy for bronchiectasis.
2. The literature on this subject has been reviewed.
3. In three reported series pulmonary tuberculosis has developed in from three to thirteen per cent of the patients.
4. All postlobectomy patients should be followed with frequent chest roentgenograms to detect the possible development of pulmonary tuberculosis.
5. When pulmonary tuberculosis is present in a patient requiring pulmonary resection for a nontuberculous disease, the surgery should be planned to avoid a situation conducive to overdistention of the tuberculous pulmonary tissue. Certain specific recommendations are given.

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# SPONTANEOUS HEMOPNEUMOTHORAX

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APPROXIMATELY eighty-four cases of spontaneous hemopneumothorax have been reported in the medical literature to date. The condition is therefore of relatively rare occurrence. The purpose of this paper is to record six more cases and to make certain suggestions regarding the treatment of the disease. One of the cases reported may be the first reported case of successful surgical intervention in the acute stage of the disease.

## HISTORICAL

Recognition of the disease was probably first made by Laennec in 1828 who found air under pressure and blood present in the pleural cavity in a case at autopsy. Whittaker, in 1876, reported the successful treatment of a case by aspiration. The first two comprehensive case reports were, however, published in 1901, independently by Pitt[1] and Rolleston.[2] Each author published a single case report of fatal spontaneous hemopneumothorax. Both cases were young men. Pitt's case of an 18-year-old boy showed at autopsy emphysematous bullae at the apex of the right lung and torn pleural adhesions. Rolleston's patient, a 21-year-old man, was admitted with a perforated peptic ulcer but, due to his extreme degree of shock, he was not operated on. He improved for a few days, but died one week after admission. Autopsy showed a right hemopneumothorax, but no apparent source of the bleeding.

Of the reported cases, approximately 25 per cent have been fatal, and it is interesting to note in the fatal cases, coming to autopsy, that there is remarkable absence of evidence of the source of the bleeding and a not uncommon finding of the presence of emphysematous bullae in the affected lung.

Hartzel[3] published an excellent paper on the subject in 1942, reviewing the literature and reporting two cases. One of his patients had suffered a spontaneous pneumothorax, with no evidence of effusion, two years previous to his admission with a spontaneous hemopneumothorax.

Since 1942 several papers on the subject have been published, bringing the number of reported cases to approximately sixty-five.

Of the number, three cases have been treated in the chronic phase of the disease by decortication,[4, 5, 6] and Hansen[6] reports an unsuccessful attempt to treat one case with a persistent fistula by thoracotomy, with intent to suture the ruptured bullae. These were too numerous to suture, and the patient later died of anoxia. There is, to my knowledge, no report of successful surgical treatment of the acute stage of the disease.

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From The Newcastle-on-Tyne Regional Thoracic Surgery Center, England.

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## INCIDENCE

The disease appears to be almost confined to males, there being only four female cases reported. This fact is difficult to explain. It has been suggested that men are more active than women and that muscular exertion is a causative factor in the rupture of a subpleural vesicle, producing the initial pneumothorax. However, this condition and its allied disorder, spontaneous pneumothorax, which is almost as common in women as in men, may appear when the patient is doing nothing more strenuous than shaving or, indeed, than reading the Sunday newspaper. It is thus doubtful if exertion is a very potent factor.

The average patient is between 20 and 40 years of age, and the right side is as frequently affected as is the left.

## PATHOLOGY

The mechanism of production of the condition is most certainly that of rupture of a subpleural bulla with the production of a spontaneous pneumothorax. Vascular adhesions are torn, and the pleural space fills with air and blood. If the fistula remains patent and if a "ball valve" mechanism is present, a tension hemopneumothorax results.

Bronchiolar scarring due to previous inflammatory disease (e.g., tuberculosis) may well be a factor in the production of subpleural valve vesicles, which, on rupture, allow the continued escape of air into the pleural cavity. The inflammatory lesions are, however, practically always healed, and pathologically the blame cannot be definitely traced to tuberculosis as the antecedent infection. Congenital cystic lung is also a cause, although, from reports, it appears to be a rather infrequent one. It may be stated that unless there is obvious gross tuberculous cavitation present in the affected lung, it is reasonably safe to assume that active tuberculosis is not a factor in the production of the disease. It is recognized that there can be exceptions to every rule of this type, and routine sputum examinations are carried out in each case.

## SYMPTOMATOLOGY

The onset may be acute and with dramatic suddenness, or it may be gradual and more insidious. In the acute stage there may be a sudden severe pain in the chest or upper abdomen, radiating to the neck and shoulder, and accompanied by syncope. There is next a latent period, in which the pain is less and the patient feels somewhat better. As the bleeding continues, shock and collapse occur. The escaping air builds up pressure in the pleural cavity and tension phenomena result. There may be nausea and vomiting due to irritation of the lower thoracic nerves with reflex via the sympathetic supply to the abdominal viscera. If the escape of blood and air is slower, the above symptoms will be less marked, and will appear over a period of hours or even days.

## DIFFERENTIAL DIAGNOSIS

One of the first cases reported was misdiagnosed as a perforated peptic ulcer, as were three of this series, and it is probable that this is a not infrequent "first diagnosis." The disease very closely resembles such acute abdominal conditions as perforated viscus, acute pancreatitis, renal or choledochal calculus. The severe chest pain with radiation to the arm and shoulder, accompanied by shock, is very suggestive of coronary occlusion. The clinical evidence of pneumothorax is, however, usually quite obvious.

## TREATMENT

It has been frequently suggested that the treatment in the acute stage of the disease, when active bleeding is present, should be that of bed rest, warmth, sedation and, if necessary, transfusion. Aspiration of the chest is advocated if the mediastinal displacement is great enough to cause respiratory embarrassment. The rationale of this treatment has been based on the belief that, as in intrapulmonary hemorrhage, rest, warmth, and sedation are beneficial, and that if the bleeding be from the lung, collapse of that organ will aid in the arrest of the hemorrhage. From observations at segmental lobectomy I would suggest that the latter part of this thesis is not entirely founded on fact, inasmuch as the bleeding tends to cease with the lung expanded.

On examination it seems that a mortality rate of 25 per cent is rather high for hemorrhage from a vessel of relatively minor caliber, and that active treatment might have been more successful than the "wait and see" attitude.

The treatment of this condition resolves itself into two phases: that of the "acute stage" and that of the "chronic stage" of the disease.

## TREATMENT IN THE ACUTE STAGE

Clinically, the acuteness of onset will vary with the rate of accumulation of air and blood in the pleural space. Generally, however, shock and dyspnea due to blood loss and mediastinal displacement are fairly well marked. There are two problems in this phase: that of blood loss and that of interference with the cardiorespiratory function by the accumulation of blood and air in the thorax. The patient should be treated as an urgent surgical emergency. Posteroanterior and lateral roentgenograms of the chest should be obtained. Blood should be matched and equipment made ready for thoracentesis and the insertion of an indwelling catheter into the chest. The intrapleural tension must be relieved and the lung allowed to expand and obliterate the pleural space. This is best accomplished by an intercostal catheter of the Malicot type, attached to a water seal, and allowed to drain gradually over a period of several minutes. Transfusion should also be started at this time. Generally speaking, the patient's condition should be now, at least temporarily, under control, and one can observe the rate at which further bleeding occurs. It may be necessary to place another catheter in the second interspace anteriorly to allow air to escape, as the presence of dependant fluid is often an effective barrier to the escape of air except under considerable pressure. The catheter in the dependent drainage position may also be blocked by the partially expanded lung or by an elevated diaphragm. Serial roentgenograms will be necessary to determine the degree of expansion of the lung. It must not be taken for granted that the lung is completely expanded, because air entry can be heard on auscultation over the affected side. It is important that every effort be made to prevent any residual space being left to require treatment at a later date. "*Now is the hour.*" The patient is nursed in the semirecumbent position unless in extreme shock, and oxygen is given as indicated.

If bleeding, as shown by the condition of the patient and the amount of drainage in the bottle, appears to be excessive and continuous, then no time should be lost in deciding upon a thoracotomy as the further "treatment of necessity." With the chest open and the bleeding point under direct vision, the problem becomes one of arrest of hemorrhage with the bleeding vessel conveniently accessible. When the space has been cleared of clot and the bleeding controlled the chest is closed, leaving two intercostal catheters attached to water seals. One tube is in the costophrenic angle and one in the second interspace anteriorly. No attempt need be made to close the fistula, if indeed it may be found, but large emphysematous bullae may be resected if the condition of the patient allows and the procedure is not too formidable. Postoperatively, every effort must be made to secure complete expansion of the lung.

Fig. 1.—(Case 1). Roentgenogram taken on admission. Note large pneumothorax, fluid level-apical adhesion. Note also the mediastinal shift to the left.

That the results of the above schedule of treatment may be expected to be favorable is illustrated by the following case.

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## TREATMENT IN THE SUBACUTE OR CHRONIC PHASE

At this time the bleeding has ceased, the fistula has usually closed, and the patient is partially adjusted to the mediastinal displacement. The problem is now one of securing re-expansion of the lung. This is complicated by the deposition of fibrin on the pleural surface as a result of defibrination of the blood in the pleural space by the motion of the heart and lungs. If the time elapsed is short (less than one week) the task is easier as the layer has not yet begun to organize. Organization proceeds rapidly from this time onward and by a month the layer contains well-formed "adult type" fibrous tissue.

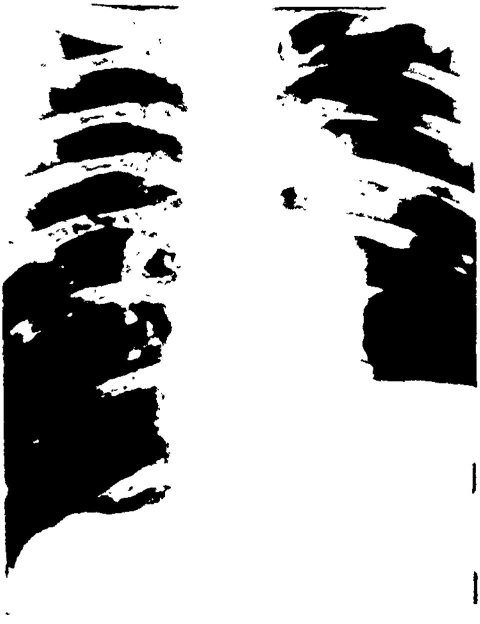

Fig. 3.

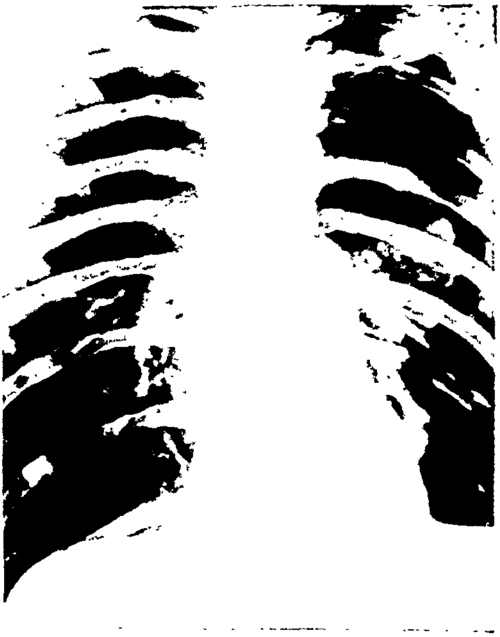

Fig. 4.

Fig. 3.—(Case 2.) Roentgenogram taken on admission, showing large pneumothorax and fluid level.

Fig. 4.—(Case 2.) Roentgenogram one month after admission. Note that the lung is still only partially expanded, and that a fluid level is still present.

In the early case one may attempt to secure re-expansion of the lung by means of repeated aspiration, saline washouts, and perhaps the use of streptokinase. This method works well, even in cases that are not so early, as is shown by the following examples.

CASE 2.—Middlesborough Chest Clinic No. 1, male laborer, aged 26 years. This man was admitted with a three-week history of pain in the left posterior chest, and gradually increasing weakness and dyspnea. He eventually became too weak to get out of bed. On admission he was quite ill, very short of breath, cyanotic, and febrile. The roentgenogram showed

CASE 1.—Shotley Bridge No. 6441, male machinist, 34 years of age. This patient was admitted with a history of having been seized with a sudden sharp pain in the right posterior chest while at work twenty-four hours previously. About sixteen hours prior to admission he had vomited undigested food, a considerable quantity of "coffee ground" material, and about a dessertspoonful of bright red blood. He had no further vomiting. He volunteered the information that he had suffered from "stomach trouble" for years. Since the onset of pain he had become somewhat short of breath, but not markedly so. He also had had several bowel movements, but no evidence of melena. On examination he was pale, breathing rapidly, but was not distressed. His pulse was 120 per minute and his blood pressure was 70 mm. systolic. One hour after admission a blood transfusion was started. The diagnosis at that time was that of gastrointestinal hemorrhage, but six hours later the patient suddenly collapsed, was cyanosed and dyspneic, and had the signs of a tension pneumothorax. Aspiration of the chest at this time yielded air and a small amount of blood, and gave immediate relief of symptoms. However, it was again necessary to aspirate the chest half an hour later, and two and one-half pints of blood were removed at this time. At this stage the patient was in extreme shock and death seemed imminent. However, by transfusing under pressure and the introduction of an intercostal catheter, he improved remarkably, and within a few hours he seemed comparatively well. Blood, however, continued to drain from the pleural space, and his condition again began to deteriorate. It was obvious that he was not going to survive unless the bleeding could be stopped. He was accordingly removed to the operating theater and a *quick* thoracotomy was performed. A large amount of clotted blood was removed from the pleural space and a partially divided adhesion was seen at the apex of the lung. In this adhesion was a small, actively bleeding artery. The opening in the artery was obviously being kept patent by the tension of the adhesions. A small bulla was also present. The division of the adhesion was completed, and the vessel was ligated. The chest was then

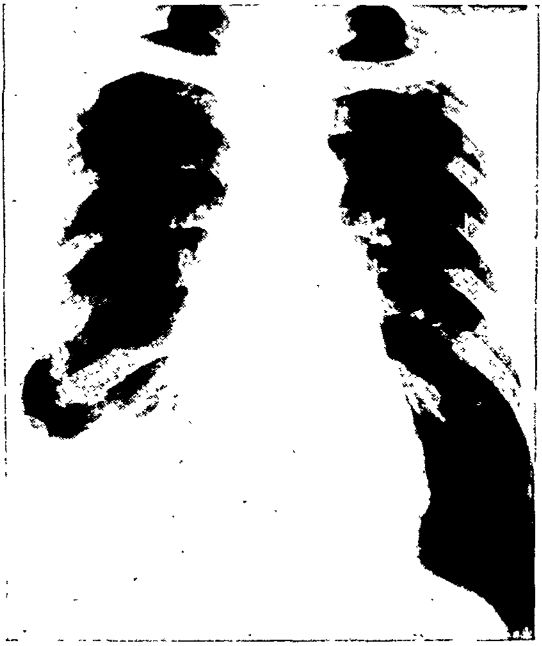

Fig. 2.—(Case 1.) Roentgenogram made at outpatient department, showing satisfactory re-expansion of the lung following thoracotomy. There are residual adhesions at the costophrenic and cardiophrenic angles.

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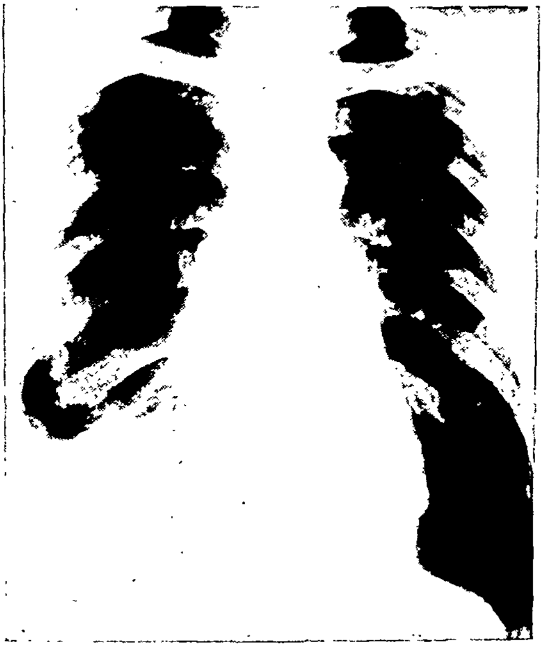

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a large left hydropneumothorax which on aspiration yielded air and dark brown fluid which was found to be old blood. Six pints of this were removed from the chest. Repeated aspirations, combined with saline washouts, resulted in complete re-expansion of the lung in three months' time.

CASE 3.—Middlesborough Chest Clinic No. 2, male post office engineer, 29 years of age. This patient developed severe pain in the left chest and shortness of breath while seated at his office desk. Later in the day he vomited repeatedly. He was treated expectantly by his doctor, and the pain gradually eased and the dyspnea became less. Three weeks later, when he had been ambulatory for about five days, he again experienced a sudden attack of chest pain and dyspnea. Chest aspiration at this time revealed the presence of a left hemopneumothorax. He was treated by repeated aspiration of the chest and re-expansion of the lung was progressive over the following six weeks.

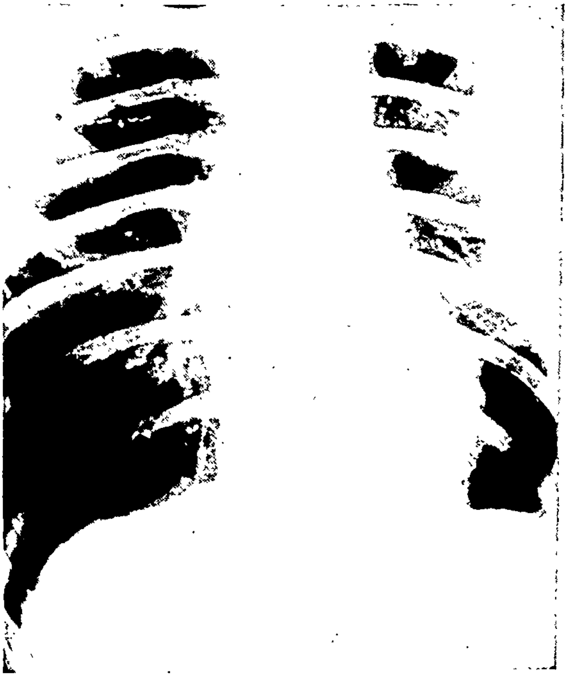

Fig. 5.—(Case 2.) Roentgenogram showing satisfactory re-expansion of the lung six months after admission.

It may be seen from the examination of these two cases that, although the results were those of complete expansion of the lung with a good functional result, the time necessary to achieve this result may be as long as three months, and it must be remembered that the risk of converting a simple clotted hemothorax into an empyema is always present.

Of the next two cases, one was first seen in the acute stage and the other in the chronic stage. Both were treated conservatively by aspiration and transfusion in the acute stage until the bleeding had ceased and the fistula was closed. In each case there remained a residual space. This was treated for a short while by further aspiration but it could be seen that cure by this

afterward 6 pints of almost pure blood were aspirated from the pleural cavity. Blood transfusion was commenced, and within a short time the patient's condition was quite good. Further aspirations were performed over the following few days. Two weeks after admission, a roentgenogram of the chest showed a partially expanded lung with multiple fluid levels intrapleurally. In this case, it is probable that if early intercostal drainage had been used instead of aspiration, the lung would have fully expanded. It was finally decided that decortication should be performed, and, following this operation, the lung re-expanded satisfactorily and the man was discharged three weeks after operation.

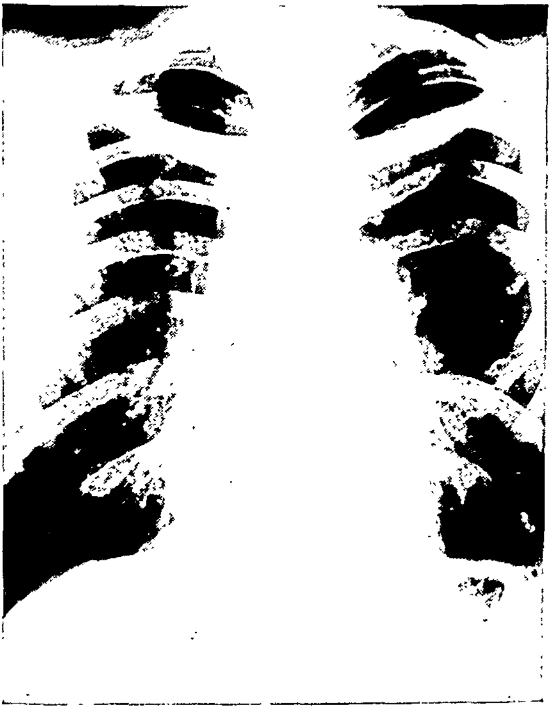

Fig. 8.—(Case 5.) Predischarge roentgenogram showing satisfactory re-expansion of the lung.

If the patient is seen at the time when organization of the fibrin layer has already begun, the treatment of choice is thoracotomy and decortication of the lung. If, even in an early case, there is a large amount of clot in the pleural space, the same rule applies.

The last case was first seen some months after the onset of his illness. He presented with a definite organizing clotted hemothorax. This man was unusual, in that he gave a long history of "chest trouble" with repeated attacks of bronchitis and "chest colds."

Case 6.—Shotley Bridge No. 6995, male shoolteacher, aged 26 years. Three months prior to admission this man developed a sudden pain in the right chest and became short of breath. He was treated as a "pleurisy" patient until his distress became so great that he had to be admitted to the hospital where a roentgenogram revealed the presence of a large right hydropneumothorax. Aspiration was performed, and 18 ounces of dark blood were obtained. As it was evident that the pleural space was filled with organizing blood clot, it was decided to transfer him to Shotley Bridge Hospital for treatment. On admission there he was a thin, pale, tired, and ill-looking young man. After transfusion and several days on a high protein diet, his condition improved considerably. It was therefore decided to decorticate his right lung. At operation a large amount of organizing blood clot was removed from the pleural space, and a moderately thick fibrous "peel" was stripped from the surface of the visceral pleura. The lung expanded well postoperatively, and subsequent bronchography showed no evidence of the bronchiectasis that had been suggested by his history. Seen recently at the outpatient clinic, he had gained approximately 30 pounds in weight since the operation, and stated that "he had not felt as well for years as he did then."

In none of the cases operated on in the chronic stage was there seen any evidence, at operation, of the cause of the pneumothorax.

## DISCUSSION

From consideration of the above cases, it is obvious that the logical treatment of this condition can be summarized as follows.

In the acute stage the procedure should be to decompress and drain the chest with an intercostal catheter attached to a water seal, transfuse, and be prepared to do a thoracotomy if necessary.

In the early chronic stage (up to one week), before organization of the fibrin has started, intercostal drainage and intensive physiotherapy should be used. If the fistula remains persistently open and the lung will not adhere to the chest wall, thoracoscopy and "poudrage" with iodized kaolin or asbestos powder is indicated. Usually, however, a pleuritis of sufficient intensity to close the fistula and produce pleural adhesion will be produced by the presence of blood in the pleural cavity.

If the case goes on to the late chronic stage without expansion of the lung being obtained, or if the patient is first seen in the late chronic stage of the disease, a thoracotomy with decortication of the lung is advised. In the case, early or late, with a large amount of clotted blood in the pleural space, it is advisable to do a thoracotomy and to clear out the clot to enable the lung to expand properly.

It has also come to my attention that an article by Myers, Johnston, and Bradshaw, reporting the successful treatment by thoracotomy of an acute case of spontaneous hemopneumothorax, appeared in the *Annals of Surgery* of March, 1949. The case reported by me is therefore not the first one.

The above additional evidence supports the suggestion that thoracotomy is a good form of treatment of selected cases of the disease.

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# THE DEVELOPMENT OF CANCER IN DYSTOPIC AND ECTOPIC THORACIC STOMACHS

## REPORT OF TWO CASES

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THE incidence of intrathoracic gastric carcinoma is low There is a paucity of medical literature on this subject. The bizarre association of gastric cancer in a partial or complete intrathoracic stomach is probably more frequent than reports would indicate for three reasons: (a) many cases are not reported, (b) the diagnoses are not accurate due to pre-existent and coexistent symptoms and radiographic abnormalities, and (c) a large proportion of the case reports are based on clinical evidence unsupported by surgical confirmation. We wish to report two instances of resectable gastric cancer occurring within the chest and characterized by the following facts: Case 1.—Laparothoracotomy and subtotal esophagogastrectomy with intrathoracic esophgogastric anastomosis; Case 2.—A remarkable instance of cancer in a thoracic stomach, treated by total gastrectomy and intrathoracic esophagoduodenostomy (first recorded instance).

*Incidence.*—The annual American death rate from cancer in general approaches 125 per 100,000 of population, and for cancer of the stomach perhaps 20 per 100,000 of population, which means an estimated 28,000 deaths yearly due to gastric cancer. Among 1,200 cases of carcinoma of the stomach directly involved in the experience of one of us (G. T. P.), there have been only two instances of its occurrence within a partial or complete intrathoracic stomach, an incidence of 1 to 600, or 0.166 per cent. The theoretical application of this ratio on a national scale would suggest that about forty-five cases of cancer in an intrathoracic segment or entire stomach should be expected yearly in the United States. As far as we can determine in a casual review of the world literature, there may not have been more than twenty to twenty-five such cancers successfully resected in all times. The ratio of cancers occurring in intrathoracically dystopic or ectopic stomachs leads to interesting speculation concerning statistical and etiologic relationships. In an experimental screening by routine gastrointestinal roentgenographic examinations of some 4,000 apparently normal American men and women over 40 years of age, Robert Sherman detected only six well-defined diaphragmatic hernias and about twenty small hiatus hernias, suggesting a ratio of approximtely 600 per 100,000 of population, a figure well below that of many reporters whose criteria are not so critical of the degree of herniation, e.g., Brick with 308 hernias found in 3,448 routine gastrointestinal roentgenographic examinations (7,500 per 100,000 of population). Utilizing the figures of Sherman, we can say from our own data that, (a) there are about 300 cases of diaphragmatic hernia existing for

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every cancer of the gastric cardia, (b) only one in sixty cancers of the gastric cardia occurs in an intrathoracic stomach, and (c) the theoretical likelihood of a patient with a diaphragmatic hernia developing cancer within the intrathoracic segment of the stomach is about one in 180,000 cases.

*Anatomic Relationships and Criteria.*—The employment of the term diaphragmatic hernia indicates that all such dystopic stomachs have an investiture of a hernial sac, in contradistinction to a false hernia or "evisceration." The phrase "hernia of the diaphragm" is not generally used in the case of the congenitally short esophagus because it suggests that the stomach was sometimes within the abdominal cavity. It would be more proper to say "thoracic stomach," but even this term implies that the entire stomach is in the chest; at least it distinguishes it from congenital absence of the diaphragm when both the stomach and intestines are in the thorax. Numerous names have been suggested for this interesting abnormality including: (1) thoracic stomach, (2) hiatus hernia with congenitally short esophagus, (3) diaphragmatic hernia of the short esophagus type, (4) partial thoracic stomach, (5) congenital shortening of the esophagus and the thoracic stomach resulting therefrom, and (6) eventration of the diaphragm (this refers to a highly placed but intact diaphragms. As far as we know, Percival Bailey first employed the term "thoracic stomach," to designate the congenital development of the stomach above the diaphragm, in which case there would be no false opening in the diaphragm, whereas in the case of diaphragmatic hernia the stomach in whole or in part may be constantly or intermittently within the chest and may be accompanied by other organs. Maurice Feldman's criteria for a true thoracic stomach are: (a) the esophagus opens into the stomach above the diaphragm, (b) no stream of barium representing the abdominal esophagus is seen to pass below the diaphragm, (c) the esophagus is not distorted or curled back on itself, and (d) the stomach remains above the level of the diaphragm and the esophagus is too short to reach the level of the diaphragm. The two case reports presented here probably represent both types; e.g., Case 1 is suggestive of cancer of the gastric cardia developing in the segment of the stomach participating in a diaphragmatic hernia (dystopic stomach); Case 2 in spite of its envelopes, is typical of a congenitally short esophagus with total intrathoracic stomach and normal diaphragmatic hiatus traversed by an attenuated duodenum.

*Etiologic Considerations.*—Can one say that a diaphragmatic hernia containing a dystopic stomach or a congenital intrathoracic (ectopic) stomach are causal factors leading to the genesis of cancer in these involved parts or should it be assumed that the two diseases are coincidental in their regional localization? The arguments may be summarized briefly:

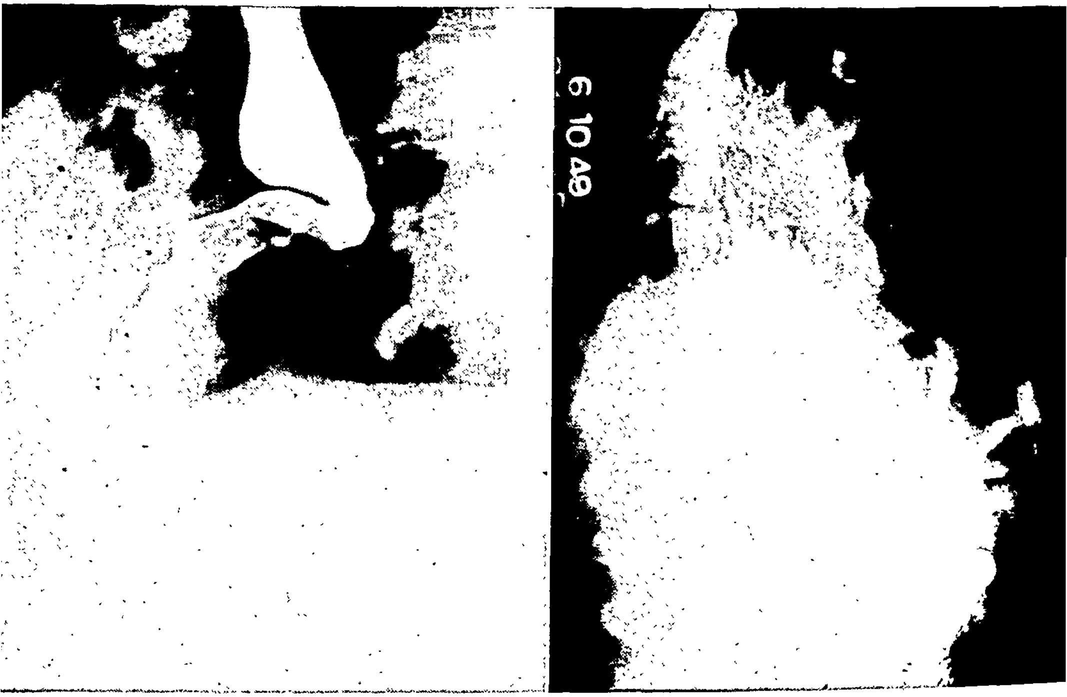
1. The previously mentioned data concerning the relative incidence of diaphragmatic hernia and gastric cancer and their coexistence in the same patient would discourage any thought of causal relationship.
2. The stomachs involved in these displacements obviously could be involved by the same diseases and with the same frequency as occurs in stomachs of otherwise normal individuals in the same age groups.

In Brick's series of 308 patients with hiatus hernias, the following associated lesions were found: duodenal ulcer, 31; esophageal diverticulum, 5; esophagitis, 3; hypertrophic gastritis, 7; gastric ulcer, 1; esophageal cancer, 1; gastric cancer, 4 (of which only two were confirmed by operation).

3. The coexistence of gastric cancer and dystopia or ectopia of the stomach should not be significant unless the cancer was situated in the herniated segment of the stomach. As a matter of fact, quite a number of the gastric cancers reported to be associated with hiatus hernias were pyloric in site (Schwartz, Jackson, and Harrington) and some of the coexistent cancers were esophageal rather than gastric in histogenesis (Hill, Wolf, Smithers, Olsen, and Harrington).
4. Do these cancers develop on ulcers occurring in the displaced or malplaced gastric segment? The assertion has been repeatedly quoted that the congenitally short esophagus is commonly associated with peptic ulceration. The common occurrence of gastric hemorrhages in patients with hiatus hernia has lent support to this theory. Nevertheless, Polley found only three gastric ulcers and no cancers in his forty-seven cases of hiatus hernia and Brick reported one gastric ulcer and four cancers in his 308 cases. In our two reported cases, the gross configurations of the cancers would render it unlikely that in their origins, they were of the ulcerocancer type.
5. Does the factor of possible irritation within the thoracic gastric segment offer a background or basis for the development of cancer in this site? With a congenitally short esophagus, the cardiac sphincter is usually relaxed which may predispose to regurgitation or retention of acid gastric juices with consequent irritation and even ulceration, although this condition should affect the esophagus more than the stomach. Patients with cancer developing in a herniated intrathoracic stomach sometimes have a long history of dyspepsia antedating the onset of the cancer (Case 2). Irritative gastritis has never been satisfactorily proved to be a precursory lesion for gastric cancer.

In conclusion, therefore, we must admit that there is insufficient valid evidence to consider dystopia or ectopia of the stomach as a precancerous state, nor is the hazard of cancer occurrence in the herniated stomach, a sufficient indication in itself to influence the surgeon's decision whether or not to repair the hernia.

*Symptomatology and Diagnosis of Cancer in the Intrathoracic Stomach.*—The resectability of gastric cancers occurring in intrathoracic segments is undeniably lower than obtains for the same disease in normally located stomachs. Indeed many of the reported cases are based on necropsy findings. The long antecedent histories of dysphagia, esophagitis, heartburn, etc., associated with the known presence of a hiatus hernia (nine years in Case 2) may lull patient and physician alike into a false sense of security. The transition in symptoms caused by the mechanical abnormality to those due to gastric cancer may be difficult to perceive. The intervention of serious hemorrhages as in one of our patients (Case 2) may lead to earlier surgical treatment. Gastroscopy with the rigid or flexible instrument may be indeterminate because of the distorted relationships of the esophagus and stomach. Correct roentgenographic diagnosis of cancer superimposed on an intrathoracic gastric segment or stomach is often difficult to make, in fact many more cases are incorrrectly diagnosed as cancer when the defect is only a mechanical distortion of a herniated portion of the stomach.



A. B.

Fig. 1.—*A* (Case 1), Roentgenogram to show supradiaphragmatic defect in gastric cardia. Note congenitally short esophagus. *B* (Case 1), Postoperative roentgenogram to show intrathoracic esophagogastrostomy after subtotal esophagogastric resection.

*Surgical Treatment.*—The more recent acceptance of laparothoracotomy as a routine measure for cancers of the proximal stomach and lower esophagus has given these patients an opportunity for cure. More successful surgical resections will undoubtedly be reported in the future. In the second case report, the total gastrectomy performed within the chest was done with the greatest ease and speed in our experience. The ligaments of the stomach and their contained blood vessels were transected and ligated with consummate ease. The duodenal stump and severed esophagus were so naturally and closely approximated that an end-to-end anastomosis well within the chest was readily done. It was not necessary to free the peritoneum along the lateral duodenolumbar gutter (Kocher maneuver) to mobilize the duodenum as was done in the olden days when subdiaphragmatic or abdominal esophagoduodenostomies were in vogue as the second phase of the operation of total gastrectomy.

## CASE REPORTS

CASE 1.—*Subtotal esophago-gastrectomy for cancer in a thoracic gastric cardia:* G. G., a white man, aged 54 years, applied for treatment on April 29, 1949. For one year he had observed increasing discomfort on swallowing solid foods. For two months he had consumed only liquids. He had recently experienced constant, dull pain in the left upper abdominal quadrant. He had previously been told that he had a peptic ulcer. Physical examination was not informative of the diagnosis.

A roentgenogram of the chest showed a density superimposed on the heart shadow. Barium swallowing and radiographic studies demonstrated a dilatation of the esophagus and a large polypoid filling defect obscuring the mucosal pattern of a dystopic proximal segment of stomach within the chest. Esophagoscopy was performed and a biopsy obtained at a distance of 40 cm. from the incisor teeth; the microscopic diagnosis was adenocarcinoma.

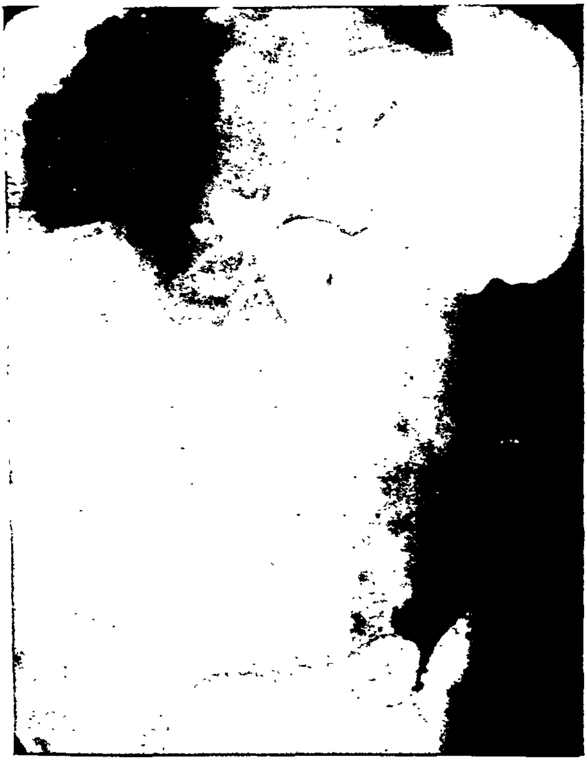
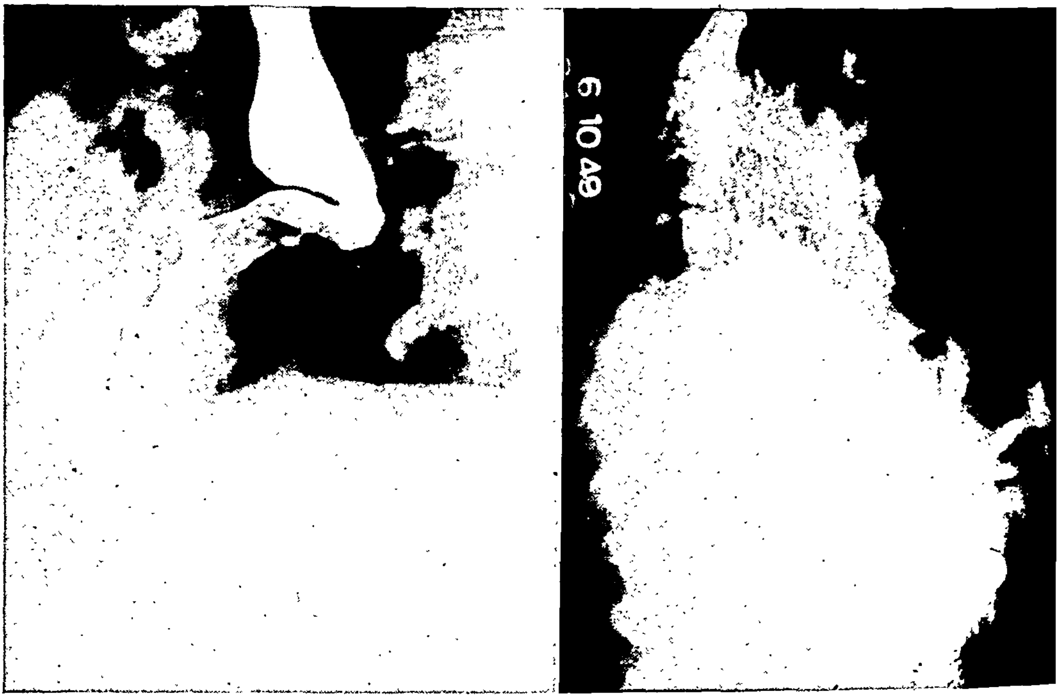


Fig. 2.—(Case 2). Roentgenogram to show intrathoracic stomach with filling defect in gastric silhouette to correspond with site of carcinoma.

A thoracolaparotomy was done on May 31, 1949. The stomach was found to be projected high within the thoracic cage. A large tumor occupied the entire gastric cardia. The brachyesophagus ended only 4 cm. below the aortic arch. Moreover, large firm lymph nodes were found throughout the omentum and along the lesser gastric curvature. The stomach was transected at the lower fifth, and the distal gastric stump was anastomosed to the esophagus immediately below the aortic arch.

The microscopic analysis was: adenocarcinoma, grade II, with extension to the peritoneum and metastases to multiple lymph nodes along the lesser curvature.

our patients (Case 2) may lead to earlier surgical treatment. Gastroscopy with the rigid or flexible instrument may be indeterminate because of the distorted relationships of the esophagus and stomach. Correct roentgenographic diagnosis of cancer superimposed on an intrathoracic gastric segment or stomach is often difficult to make, in fact many more cases are incorrectly diagnosed as cancer when the defect is only a mechanical distortion of a herniated portion of the stomach.



*A.* *B.*

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*Surgical Treatment.*—The more recent acceptance of laparothoracotomy as a routine measure for cancers of the proximal stomach and lower esophagus has given these patients an opportunity for cure. More successful surgical resections will undoubtedly be reported in the future. In the second case report, the total gastrectomy performed within the chest was done with the greatest ease and speed in our experience. The ligaments of the stomach and their contained blood vessels were transected and ligated with consummate ease. The duodenal stump and severed esophagus were so naturally and closely approximated that an end-to-end anastomosis well within the chest was readily done. It was not necessary to free the peritoneum along the lateral duodenolumbar gutter (Kocher maneuver) to mobilize the duodenum as was done in the olden days when subdiaphragmatic or abdominal esophagoduodenostomies were in vogue as the second phase of the operation of total gastrectomy.

carcinoma. An esophagoscopy was done, and a granular lesion visualized which was interpreted as carcinoma. A biopsy was performed which was not confirmatory of the clinical and radiographic opinion.

On April 14, 1950, a laparothoracotomy was done. From the abdominal aspect the stomach was absent, being retracted into the chest, with the greatly elongated duodenum extending into the diaphragmatic hiatus. The intrathoracic stomach was covered by a two-layered membrane, presumably peritoneum and mediastinal pleura, between which was a moderate serous transudate. A bulky tumor occupied the middle third of the stomach, and was situated chiefly on the lesser curvature. The tumor measured 6 cm. in diameter and invaded all coats of the stomach wall. The great omentum which was also within the chest contained numerous lymph nodes measuring 0.5 to 2.0 cm. in diameter.

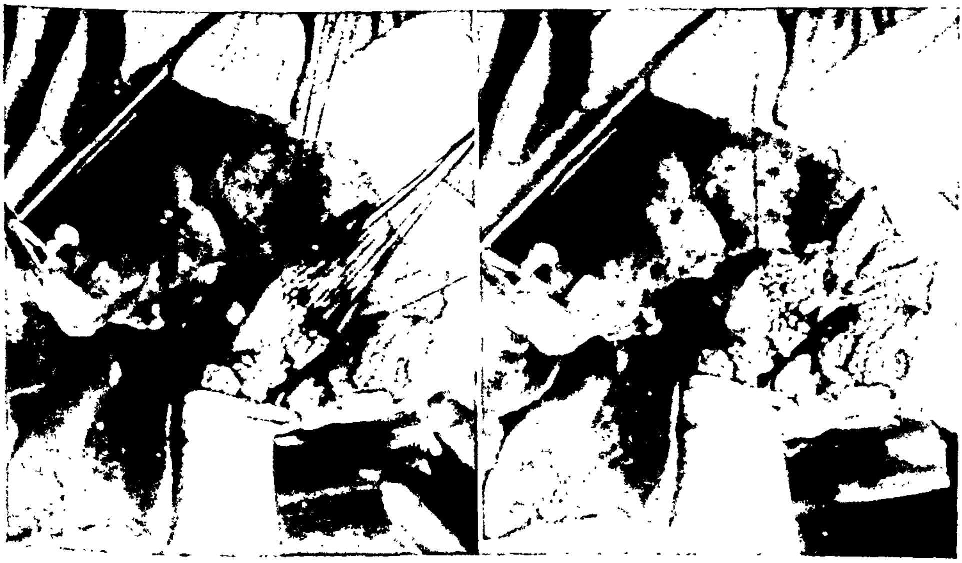

Fig. 4.—A (Case 2). Visceral view through laparothoracotomy incision after total gastrectomy. Photograph illustrates stage in esophagoduodenal anastomosis. B (Case 2). Completion of intrathoracic esophagoduodenostomy. The liver, lungs, spleen and pericardial fat have been retracted to afford a better view.

Convalescence was normal, and he was discharged from the hospital on the twenty-third postoperative day. He achieved a good appetite and was able to ingest a high-protein, high-caloric, normal diet. On March 15, 1950, his liver was found to be massive, hard, and nodular. He died on April 11, 1950, one year following the gastrectomy and intrathoracic anastomosis.

The final diagnosis was adenocarcinoma occurring in an intrathoracic cardiac segment of the stomach associated with a congenitally short esophagus.

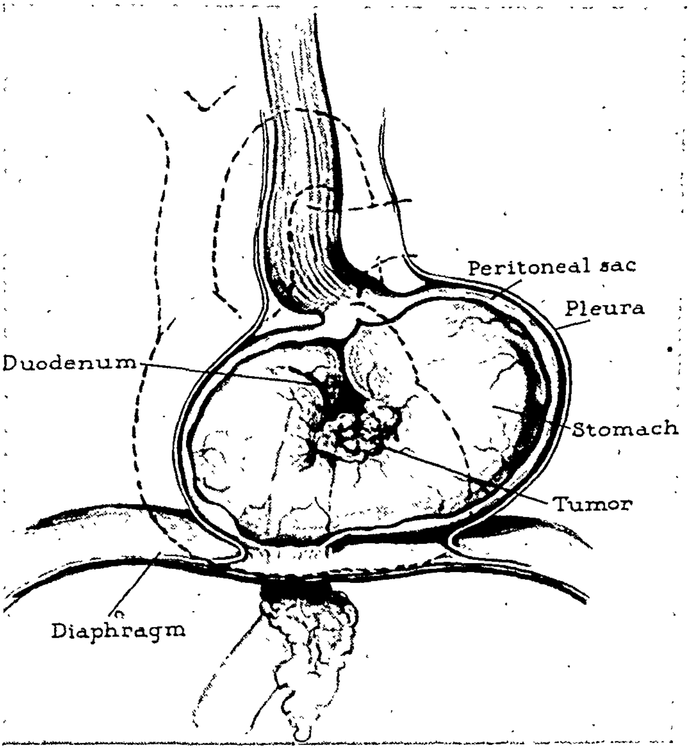

Fig. 3.—(Case 2). Diagram to show the relations of the cancer-bearing intrathoracic stomach. Note the proximity of duodenal bulb to the distal esophagus and the double investiture by peritoneal and pleural sacs.

Case 2.—*Total gastrectomy for cancer occurring in an intrathoracic stomach: intrathoracic esophago-duodenostomy:* M. G., a white man, aged 63 years, applied for treatment on April 6, 1950. His chief complaints were belching, eructation of liquids, postprandial pain, weakness, and loss of weight (40 pounds) of three months' duration. Another physician had treated him for anemia. On several occasions he had hemorrhages (hematemesis and melena), and had received six blood transfusions.

In his past experience he had occasional indigestion for many years: nine years previously roentgenographic studies were made following a barium swallowing, at which time the patient had been informed that his entire stomach was within his chest.

On physical examination, the patient appeared very anemic and cachectic. No other external evidence of disease was seen, and there were no palpable masses. Roentgenographic studies at this time revealed the stomach to be situated within the left hemithorax. There was a marked irregularity of the gastric silhouette, strongly suggesting the presence of a

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the pyloric ring. Microscopic study showed diffuse invasive replacement of the gastric wall by adenocarcinoma. The cells were of the signet-ring type. None of the perigastric lymph nodes contained metastatic cancer.

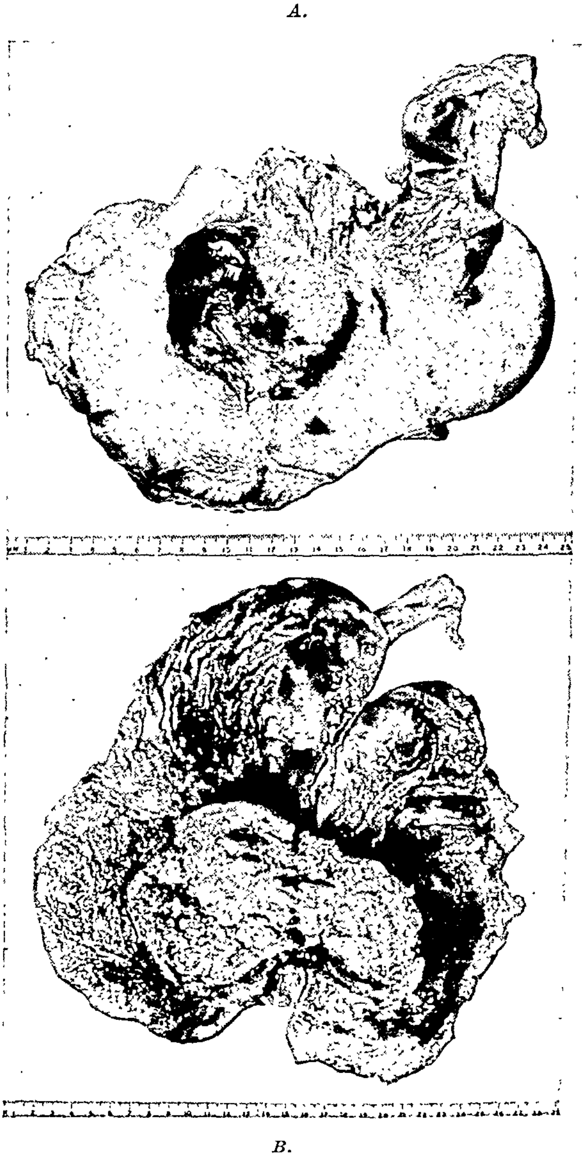

Fig. 5.—*A* (Case 2). Gross surgical specimen showing limits of duodenal and esophageal resection. *B* (Case 2), Ulcerating fungating saddle carcinoma developing on lesser curvature.

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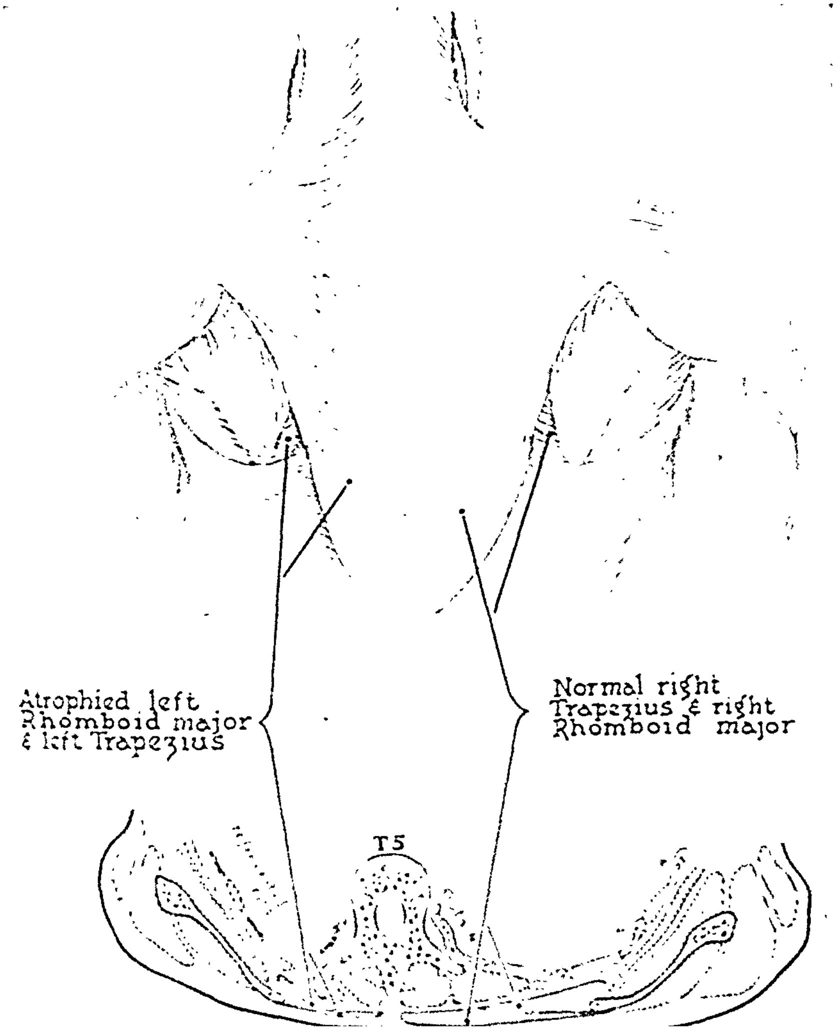
Following the thoracoplasty there is always a relative atrophy of the musculature of the operated side (Fig. 1). There is an atrophy of the trapezius and rhomboid muscles on this side. There is also an atrophy of some of the flat and long muscles of the back, as can be seen in the cross section (Fig. 1).



# SURGICAL TREATMENT OF POST-THORACOPLASTY PAINFUL BACK

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SAN ANTONIO, TEXAS

APPROXIMATELY two and one-half years ago failure to relieve a patient of post-thoracoplasty pain by resection of the inferior angle and part of the body of the scapula resulted in a careful re-examination and evaluation of the *location* of the pain and of the *type of motion* that would increase it. It was found that the cause of the pain was the impingement of the scapula upon the vertebral column. Occasionally in any large series of thoracoplasties, there are some patients who, in spite of adequate physiotherapy, training in muscle education, and proper posture, complain of almost constant pain in their backs. Some with limited thoracoplasties are found to have as the cause of discomfort or pain the familiar condition of the inferior angle of the scapula impinging upon or snapping over the last remaining rib. This report does *not* concern itself with this group, for when careful examination reveals this condition to be present, resection of the required amount of the body of the scapula and/or resection of a portion of the next remaining rib usually suffice to relieve the symptoms. Surgeons are sufficiently aware of this anatomic situation to include frequently partial resection of the scapula as part of many limited thoracoplasties.

There are, however, patients whose cause of pain has no relation to this type of condition. This is quite evident, for there are patients whose pain persists who have previously had a resection of the inferior angle of the scapula, as in the case to be presented. Careful examination will reveal that the cause of their discomfort is the approximation or overriding of the vertebral border of the scapula to or onto the vertebral column. They have a constant dull ache in their backs or are aware of a sense of discomfort at all times, and this becomes pain whenever they "brace up" in an attempt to improve their posture or whenever they reach backward in such a manner as to push their scapula backward. All the patients complaining of this pain clearly list inability to rest or sleep on the side of the thoracoplasty. It is important in examining the patient to find out whether the pain radiates in such a fashion that one might feel that the intercostal nerves are involved. These patients do not have the deep intercostal nerve pain, but state their pain is in the paravertebral area and does not radiate.

Careful examination of these patients reveals how readily the malposition of the scapula occurs. When the patient braces, pulling the shoulder backward, one can see and feel the approximation or overriding. The jamming of the scapula against the vertebral column may be of sufficient tightness almost to hold in situ a sheet of paper.

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Following the thoracoplasty there is always a relative atrophy of the musculature of the operated side (Fig. 1). There is an atrophy of the trapezius and rhomboid muscles on this side. There is also an atrophy of some of the flat and long muscles of the back, as can be seen in the cross section (Fig. 1).

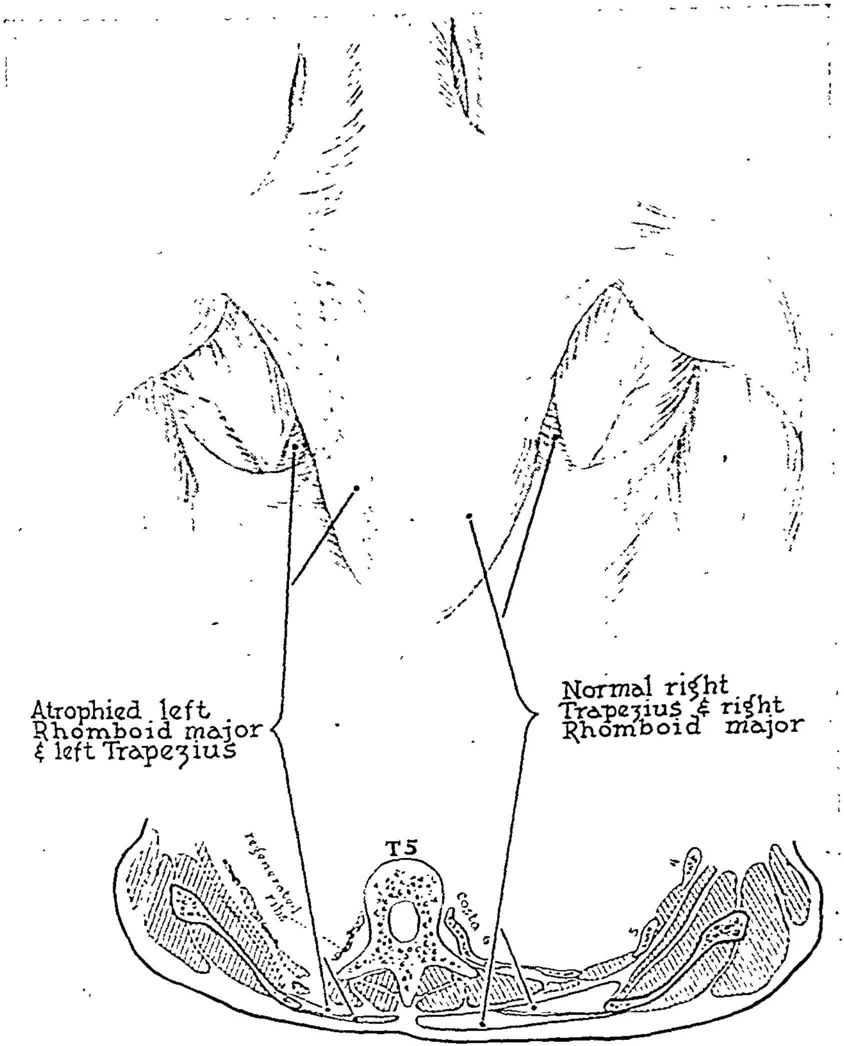

Fig. 1.—The atrophy following a thoracoplasty is depicted above. In the cross section the transverse processes are shown in order to maintain the relationships.

The following may be involved all or in part: Longissimus dorsi and cervicis, spinalis and semispinalis dorsi, and multifidus. These are the muscles that pad in the concavity between the dorsal spines and the transverse processes. This atrophy results from the stretching of this muscle group due to the scoliosis following a modern radical thoracoplasty. The unopposed action of the ligaments and muscles on the contralateral side acts as a bowstring. This scoliosis is, of course, less marked when the transverse processes are not disturbed, but the condition being described can occur following a thoracoplasty wherein the transverse processes are not disturbed, as in one of the cases. The vertebral column loses its protective padding by such atrophy. This atrophy can be noticed if one observes this region in the process of exposure for resections under previous thoracoplasties. It permits the scapula in the backward movement of the shoulder to ride against or even onto the vertebral column. There is also in any given patient the possibility of replacement of the trapezius and rhomboid muscles by a thin layer of scar tissue. This can be expected in those pa-

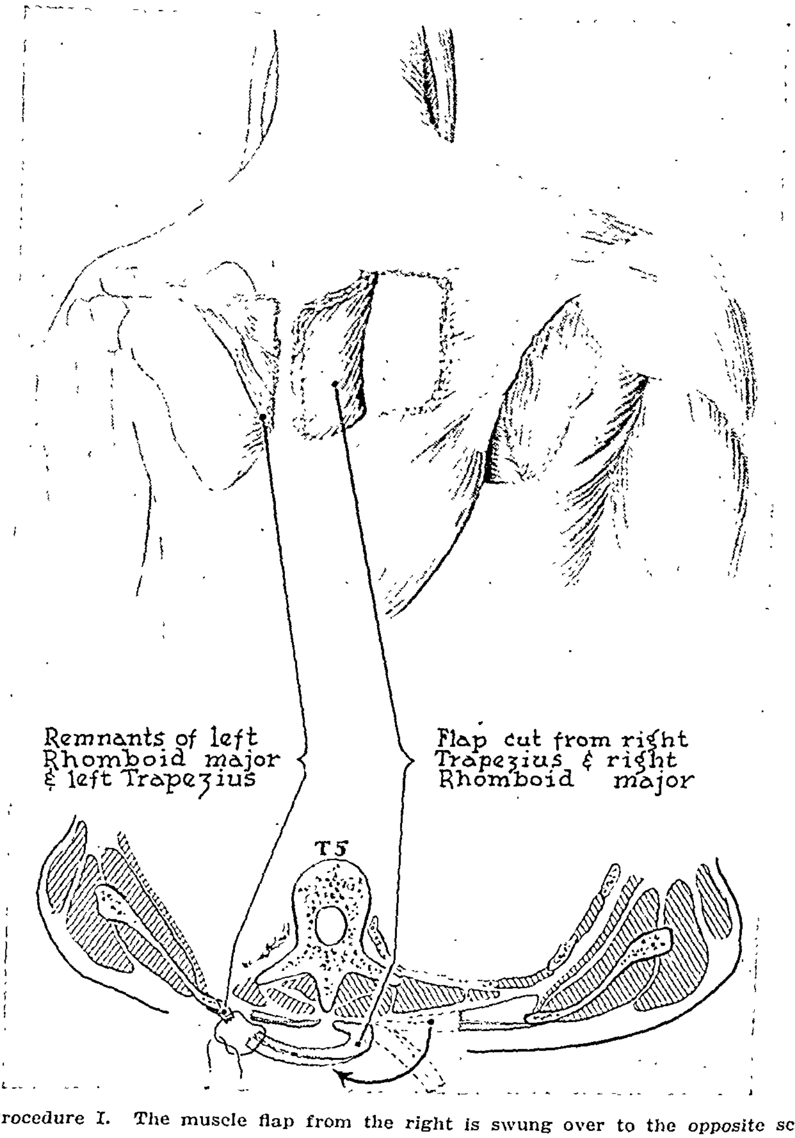

Fig. 2.—Procedure I. The muscle flap from the right is swung over to the opposite scapula.

tients in whom there occurs the following during the thoracoplasty stages: inadequate primary closure of the muscle layers, infection with greater or less disruption of the wound, and excessive fluid formation that is not immediately recognized and removed. The latter can disrupt muscle layers and be contained by the skin closure. In the eight patients who comprise this series, attempts were made to determine which, if any, of these conditions were present at the time of thoracoplasty. As the thoracoplasties in seven of the patients were not done by the author, it was difficult to elicit the above complications. However,

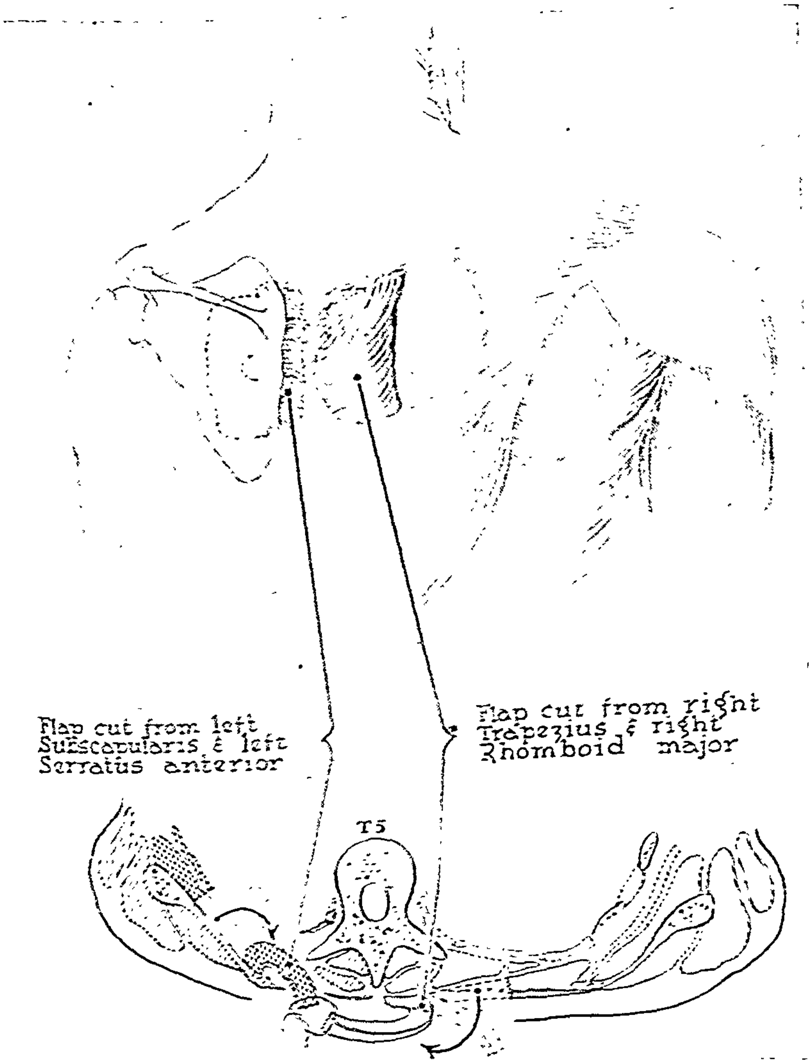

Fig. 3.—Procedure II. The muscle flap from the right and a flap from the scapula are obtained as shown above.

in at least one of the seven, infection may well have been a factor. The eighth case was due to neglect in aspiration of a collection of subscapular fluid, wherein the muscle was disrupted and the fluid was contained by the skin closure.

## OPERATIVE PROCEDURES

The bases of the treatment of this condition are operative procedures designed to provide a pad between the vertebral border of the scapula and the dorsal vertebrae. Two procedures have been used. The procedure of choice in a given patient depends upon the amount and thickness of the muscle found remaining on the vertebral border of the scapula of the thoracoplasty side.

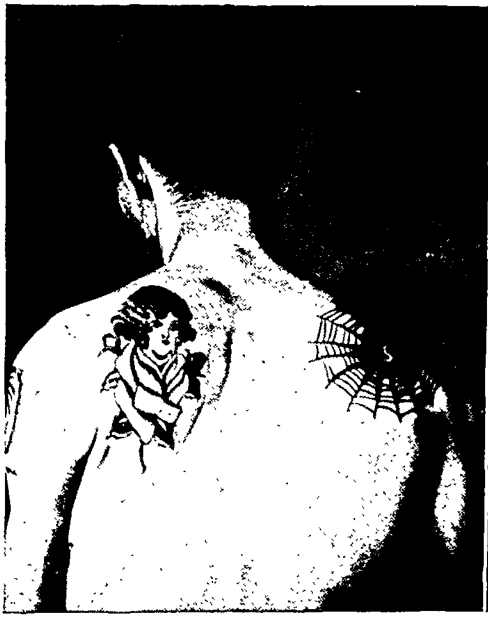

Fig. 4.

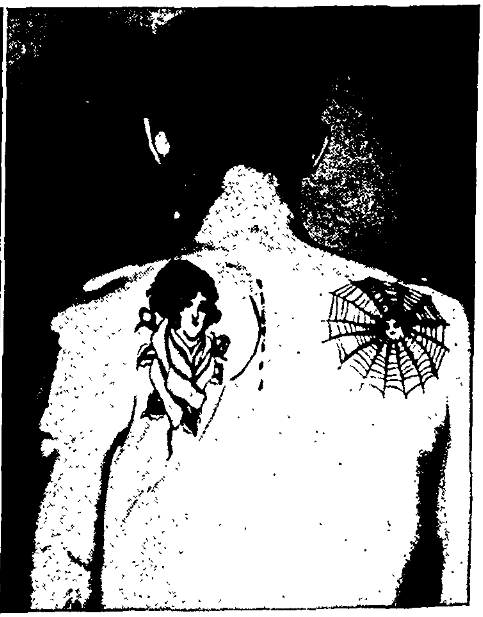

Fig. 5.

Fig. 4.—Preoperative—The impingement of the scapula against the vertebral column is shown above with the patient sitting in normal position. The atrophy of the muscles is demonstrated by the depression medial to the vertebral border of the left scapula.

Fig. 5.—Preoperative—The vertebral border of the left scapula is outlined by a solid line and the dorsal spines are marked. The overriding is clearly demonstrated.

Procedure I (Fig. 2) consists of raising a flap of trapezius and rhomboid muscle from the *opposite* side of the chest, turning the flap on itself, and attaching its free edge to the muscle remaining on the vertebral border of the scapula on the side of the thoracoplasty. Exposure and mobilization are obtained as follows: the greater part of the scar of the previous thoracoplasty operation is removed. The skin and subcutaneous tissue nearest the vertebral column is undercut and dissected *across* the dorsal spines over to the edge of the vertebral border of the opposite scapula. A muscle flap of trapezius and rhomboid is cut from this side, based on their attachments to the vertebral column. This flap is then turned on itself across the dorsal spine (as seen in the cross section, Fig. 2) and attached to the muscle remnants along the vertebral border of the scapula. In preparing the muscle along the vertebral border of the scapula of the thoracoplasty side, the scar tissue replacement can be incised, and some fairly good muscle fibers may be found and thus utilized.

Procedure II (Fig. 3): Exposure is obtained and the muscle flap raised from the opposite side as in Procedure I. When, after careful dissection, insufficient muscle is found along the vertebral border of the scapula of the thoracoplasty side, a flap may be raised from the undersurface of the scapula, consisting of the subscapularis and serratus anterior muscles. This flap is based on the vertebral border of the scapula and is turned on itself (as seen in the cross section, Fig. 3) so that its free edge may be attached to the free edge of the flap from the opposite side.

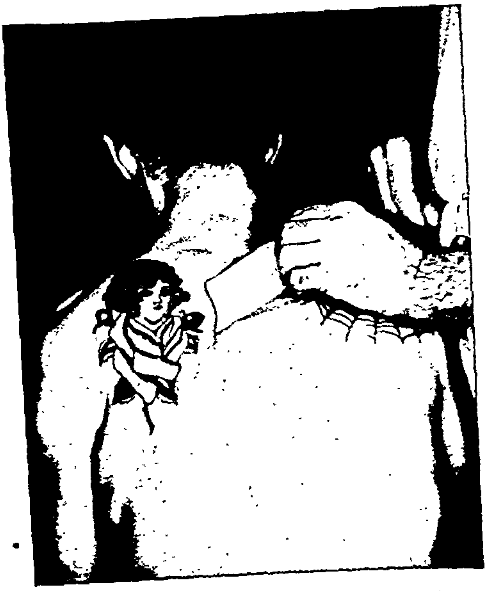

Fig. 6.—Preoperative—The overriding of the left scapula upon the vertebral column is in this case almost sufficient to hold a piece of paper in situ.

In both procedures, the regions of the source of each flap and its eventual point of suture are drained for twenty-four hours with flat rubber drains.

POSTOPERATIVE CARE

A pressure dressing is applied to the area of surgery and the arm is strapped to the side. The shoulder is partially pulled backward and held by adhesive running from the shoulder diagonally down and across the operative area onto the opposite chest. The skin sutures and pressure dressing are removed on the sixth day, the arm remaining fastened to the patient's side for ten days. Physiotherapy is not started until the third or fourth week.

A typical example is as follows: A. L. A., a white man, aged 48 years, with pulmonary tuberculosis diagnosed in May, 1945, as moderately advanced and active. Initial left pneumothorax was unsuccessful in April, 1949, and a left six-rib thoracoplasty done elsewhere was completed in July, 1949. Roentgenograms revealed excellent collapse with adequate removal of each rib and transverse process. The inferior angle of the scapula was resected as part of the second stage. Sputum and gastric cultures were negative for tuberculosis in July, 1950. Starting approximately one month after completion of the operation, the patient had discomfort on attempting to pull his shoulder back. In three more months this progressed to actual pain on attempting to reach behind him with his left hand. He had never been able to lie or sleep on his left side since the thoracoplasty. Fig. 4, 5, and 6 demonstrate the preoperative findings. Fig. 4 shows the impingement of the scapula onto the vertebral column with the patient sitting in normal position. The atrophy of the muscles is demonstrated by the depression medial to the vertebral border of the left scapula. In Fig. 5 the overriding is clearly demonstrated, as the dorsal spines and scapula are outlined. Fig. 6 reveals that the overriding of the left scapula upon the vertebral column is in this case sufficient to hold a piece of paper in situ. Figs. 7, 8, 9, and 10 demonstrate the result approximately one and one-half months after surgical Procedure II was carried out. Fig. 7 shows the result with the patient's arms pulled forward. The fullness of the left paravertebral area can be observed, as well as a slight swelling over the dorsal spines at the point of crossing of the muscle flap from the right. Fig. 8 indicates the relative positions of the medial border of the scapula and the dorsal spines with the patient sitting in normal position. Fig. 9 shows the bulge of the interposed muscle flaps when the shoulder is pulled backward. Fig. 10 demonstrates the relative positions of the vertebral border of the scapula and the dorsal spines when the shoulder is pulled backward.

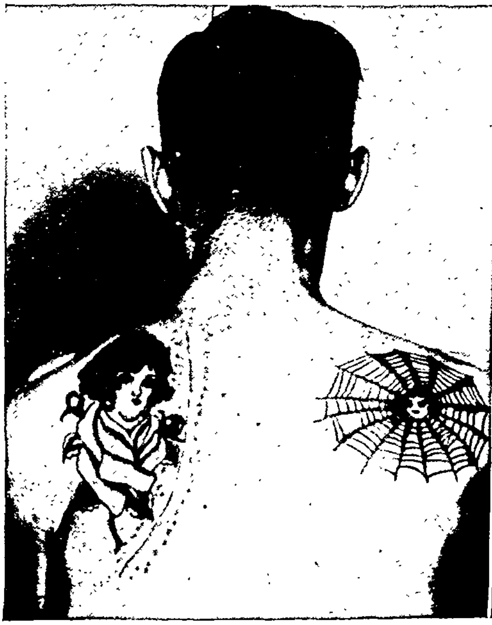

Fig. 7.

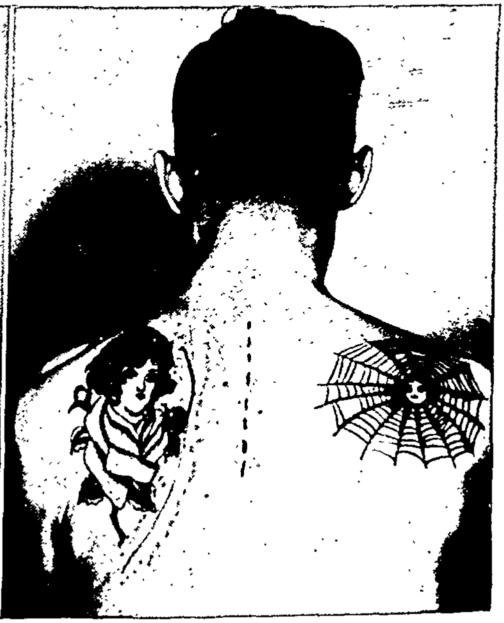

Fig. 8.

Fig. 7.—Postoperative—Patient with arms pulled forward. The fullness of the left paravertebral area can be observed, as well as a slight swelling over the dorsal spines at the point of crossing of the muscle flap from the right.

Fig. 8.—Postoperative—This indicates the relative positions of the medial border of the scapula and the dorsal spines.

## RESULTS

Eight patients have been operated upon by Procedures I and II in the last two and one-half years, and all were relieved of their symptoms. The first one was operated on in May, 1949, and the last in March, 1951. There has been no atrophy of the muscle flaps. There has been no interference in range of motion not previously present. Procedure I was done in five patients, and Procedure II was necessary in the remaining three. Three patients originally had a partial scapulectomy. Two of these scapulectomies were done as part of a second stage thoracoplasty, and one was done following the last stage of thoracoplasty. Five of the thoracoplasties were done in three stages, and three in two stages. The

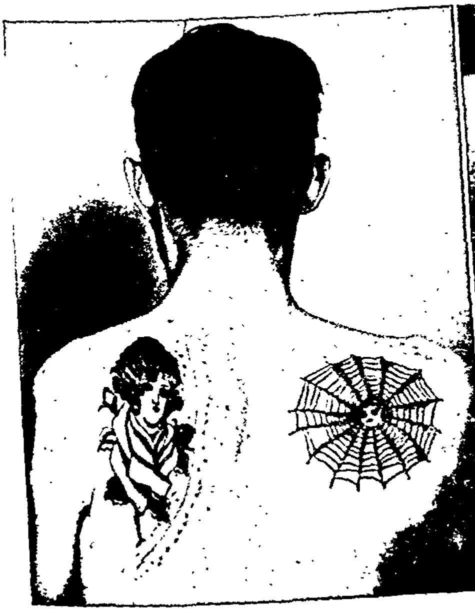

Fig. 9.

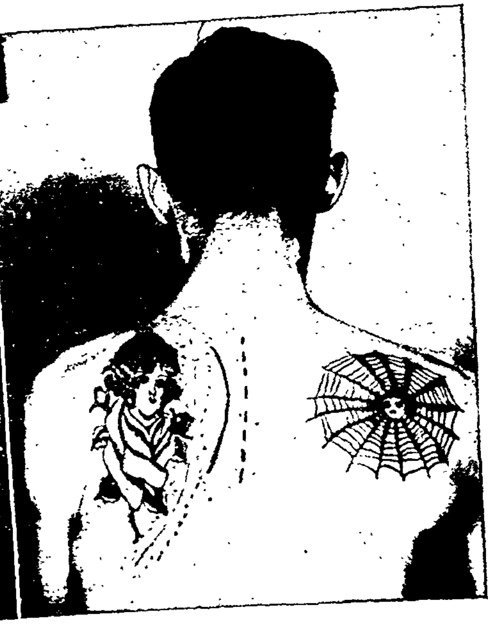

Fig. 10.

Fig. 9.—Postoperative—This indicates the bulge of the interposed muscle flaps when the shoulder is pulled backward.

Fig. 10.—Postoperative—This demonstrates the relative positions of the vertebral border of the scapula and the dorsal spines when the shoulder is pulled backward.

most ribs removed in one case were nine, and the least were five. In all but one, the posterior rib segments and transverse processes were removed. The interval of time between the last stage of thoracoplasty and surgical Procedures I and II averaged fourteen-plus months—the longest was twenty-four months and the shortest eight months.

## DISCUSSION

When one sees large groups of thoracoplasty patients, it becomes obvious that many of them have aches or pains of one sort or another with reference to their thoracoplasties. It is apparent that it requires careful, patient, and detailed examination in order to find out whether these symptoms are due to the more common cause of the impingement of the inferior angle of the scapula over remaining ribs, true intercostal nerve pain, or, less commonly, the anatomic condition described in this report. It is advised that each patient complaining of pain be carefully examined and, without asking leading questions, be permitted to tell you where his pain is and what brings it about. It is felt that patients who could receive marked or complete relief by the procedures described herein are passed by with too casual an examination. This may be observed in the fact that in one of the patients, resection of a portion of the scapula was done to relieve his pain and it failed completely, there being no change in the patient's symptoms after the partial scapulectomy. When re-examined, the findings and history revealed the condition to be present as described herein, and one of the surgical procedures gave him complete relief. It is obvious that only inadequate examination and questioning resulted in some unnecessary surgery.

## CONCLUSIONS

1. Attention is called to the post-thoracoplasty impingement or overriding of the scapula onto the vertebral column as the cause of pain.
2. Possible causes in development of this condition are listed.
3. Two operative procedures used in alleviation of this condition are described.
4. Results in eight cases are reported, and a case is presented demonstrating the pre- and postoperative condition.

# LIMITED THORACOPLASTIES WITH EXTRAFASCIAL APICOLYSIS

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SINCE the advent of thoracoplasty in the treatment of pulmonary tuberculosis in 1888, modifications of the original procedure have been legion. In recent years, however, the "standard thoracoplasty" in the United States has been that advocated and popularized by Alexander and Churchill and their followers. Despite good general results reported in the literature, there have been from 15 to 25 per cent failures, the deformity has been considerable, and there has been immobilization of rather large portions of adjacent normal lung because of the extent of the rib resection. Little known and poorly understood in this country is Semb's apicolysis used in conjunction with a limited thoracoplasty.[1] The applicability and success of this method will be illustrated in the following series of consecutive thoracoplasties of five or less ribs performed in the Oslo Municipal Hospital and the Oslo Municipal Tuberculosis Sanatorium from 1942 to 1949.

There are 174 cases in all, five bilateral, with a total of 261 stages of operation. The operations were done according to the method of Semb,[*] many of them performed by him.

Since the development of this method in 1935, the operation has become more moderate and selective. Fewer and less extensive segments of ribs are resected without reducing the size of the apicolysis. Thus a seven-rib thoracoplasty has given way to a six-rib combined with partial scapulectomy, to a five-rib two-stage procedure, to a one-stage four-rib operation. The results have not suffered from this reduction in the extent of the rib resection.

Another modification has been the development of a method of maintaining the collapse after operation. In the early years up to 1944, only saline was placed in the apicolysis cavity. Then mineral oil filling was begun with noteworthy success in preventing re-expansion of the apex until the ribs had regenerated over the new apical level. In an effort toward further improvement, air filling[†] was instituted in 1947, introducing pressures up to 40 to 50 cm. of water. These fillings are done on the fourth, eighth, and twelfth postoperative

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[*] Through a paravertebral subscapular incision about one-third of the third rib, three-fourths of the second, and all of the first rib are resected dividing their intercostal bundles. The apex of the lung is freed in the extrafascial plane beginning in the sheath of the brachial plexus and extending down beyond the azygos vein on the right and the arch of the aorta on the left: anteriorly to the second rib and posteriorly to the sixth or seventh ribs. The space is half filled with saline before closure. At the second stage the space is reopened; about one-half to two-thirds of the fourth, one-third of the fifth, the remainder of the second and most of the residual third ribs are resected. The intercostal bundles are divided posteriorly and often sutured over the new apical level. Cancellous bone chips are also in recent years placed over the apex to supplement the regenerated ribs. The space is again half filled with saline followed by postoperative air filling (Fig. 1.).

[†] An ordinary pneumothorax needle is inserted at the upper end of the incision toward the nipple in order to enter the upper posterior part of the apicolysis space. When the manometer moves with respiration or coughing, the cavity has been reached. Air is then insufflated up to pressures between 20 to 30 c.c. of water. Later following the second stage of operation pressures can be effected up to 60 c.c.

days after the first stage of the operation, fourth, tenth postoperative days after the second stage, and weekly thereafter until new ribs are seen and the new thoracic roof is stable. The residual air is then gradually absorbed.

Of the 174 cases, 93 were five-rib, 66 were four-rib, and 15 were three-rib thoracoplasties. This final group was composed primarily of cases with postoperative complications or x-ray evidence of cavity closure. The over-all mortality was 3.5 per cent, and rate of cure 88.5 per cent (Table I).

Of the five-rib group eighty-two, or 88 per cent, of the ninety-three cases have no roentgenographic evidence of cavitation, as evidenced by planigrams, are repeatedly negative by culture, and are in full- or part-time work. The operative mortality in this group is 1.1 per cent, and there is a late mortality of 2.2 per cent. The residual cavity rate is 8.6 per cent.

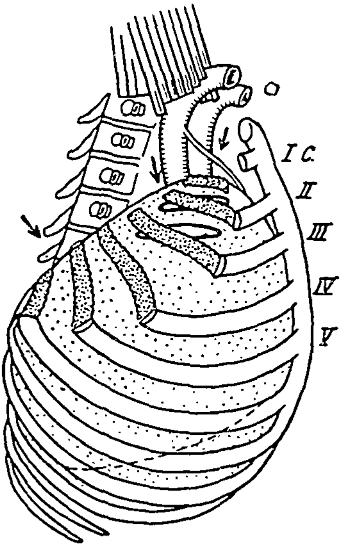

Fig. 1.—Diagrammatic representation of the extent of rib resection in a six-rib thoracoplasty.

TABLE I. COMBINED STATISTICS OF CONSECUTIVE CASES FROM 1942 TO 1949. SMALL THORACOPLASTIES

| NO. RIBS | NO. PAT. | STAGES | NO. BILATERAL OP. | INFECTION | MORTALITY LESS THAN 2 MO. | LATER MORTALITY | FOLLOW UP 3 MO. TO 7 YR. | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | CAV.- TB.- | CAV.+ TB.+ |
| 3 | 15 | 15 | - | 2<br>13 per cent | 1<br>6.6 per cent | 3<br>20 per cent | 10<br>66.6 per cent | 1<br>6.6 per cent |
| 4 | 66 | 74 | 2 | 1<br>1.5 per cent | 1<br>1.5 per cent | 1<br>1.5 per cent | 62<br>94 per cent | 2<br>3.3 per cent |
| 5 | 93 | 172 | 3 | 5<br>5.4 per cent | 1<br>1.1 per cent | 2<br>2.2 per cent | 82<br>88 per cent | 8<br>8.6 per cent |
| Total<br>Per cent | 174 | 261 | 5 | 8<br>2.2 per cent | 3<br>1.7 per cent | 6<br>3.5 per cent | 154<br>88.5 per cent | 11<br>6.3 per cent |

Of the sixty-four four-rib cases, sixty-two, or 94 per cent, have no roentgenographic evidence of cavity, and are repeatedly negative on culture. The operative mortality and late mortality is 1.5 per cent. The residual cavity rate is 3.3 per cent.

The three-rib group of fifteen cases resulted in ten cures or 66.6 per cent. The operative mortality was 6.6 per cent, and late mortality 20 per cent. Residual cavity has been found in one, or 6.6 per cent.

The combined late mortality in the four- and five-rib thoracoplasties was 1.9 per cent.

The follow-up period is from under one year to seven years, with about half of the cases two or more years (Table II).

The cavities have been located predominantly above the level of the fifth rib posteriorly (Fig. 2). They can be classified in three groups: Group I, 0 to 2.5 cm.; Group II, 2.5 to 4 cm.; and Group III, 4 to 5 cm. The greatest number were in the first group (Table III).

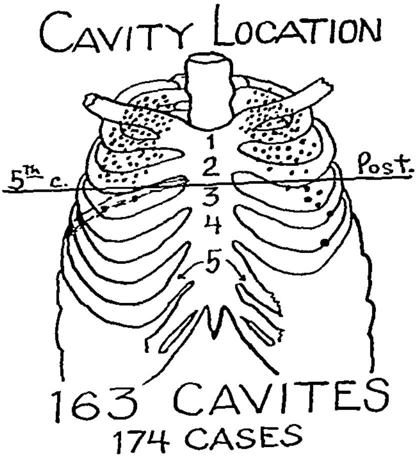

Fig. 2.—Cavity location

TABLE II. OBSERVATION TIME

| RIBS | UNDER & 1 YR. | 2 YR. | 3 YR. | 4 YR. | 5 YR. | 6 YR. | 7 YR. | TOTAL |
|---|---|---|---|---|---|---|---|---|
| 3 | 6 | 3 | 5 | – | 1 | – | – | 15 |
| 4 | 41 | 9 | 5 | 7 | 2 | 2 | – | 66 |
| 5 | 44 | 18 | 12 | 9 | 7 | 2 | 1 | 93 |
| Total | 91 | 30 | 22 | 16 | 10 | 4 | 1 | 174 |

TABLE III. CAVITY SIZE ACCORDING TO EXTENT OF RIB RESECTION

| Ribs | I 2.5 CM. | II 2.5-4 CM. | III 4-5 CM. | SINGLE | MULT. | TOTAL |
|---|---|---|---|---|---|---|
| 3 | 15 | – | – | 14 | 1 | 15 |
| 4 | 58 | 3 | – | 54 | 7 | 61 |
| 5 | 69 | 15 | 3 | 67 | 20 | 87 |
| Total | 142 | 18 | 3 | 135 | 28 | 163 |

When the cavity size is plotted against the number of ribs resected, it is found that of 142 cases from Group I (0 to 2.5), 129 or 90.8 per cent were treated successfully, one died in the postoperative period, and seven, or 4.9 per cent, were left with residual cavities. This contrasts sharply with the next group (2.5 to 4) where of the eighteen cases, although there were no operative deaths, only thirteen or 72.2 per cent were cured, and four or 22.2 per cent had residual cavities. This suggests that cavity size is an important factor in determining the chance of success with a small thoracoplasty (Table IV).

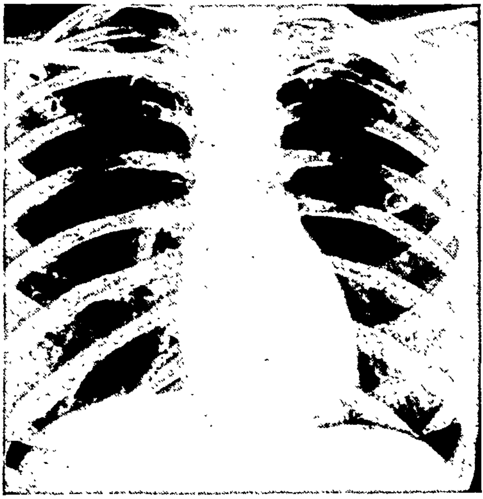

Fig. 3.

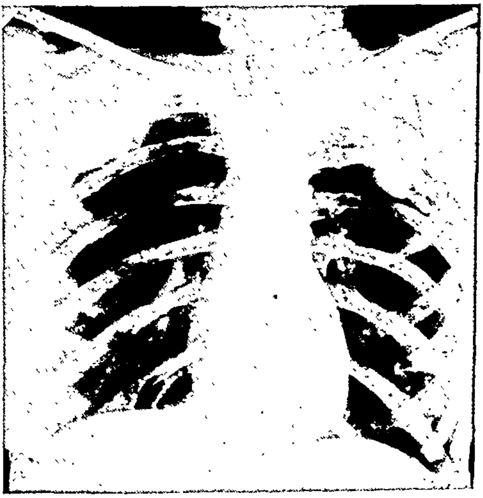

Fig. 4.

Fig. 3.—(Case 1.) Preoperative roentgenogram.

Fig. 4.—(Case 1.) Eight months after the four-rib thoracoplasty on the left and four *months after that on the right.*

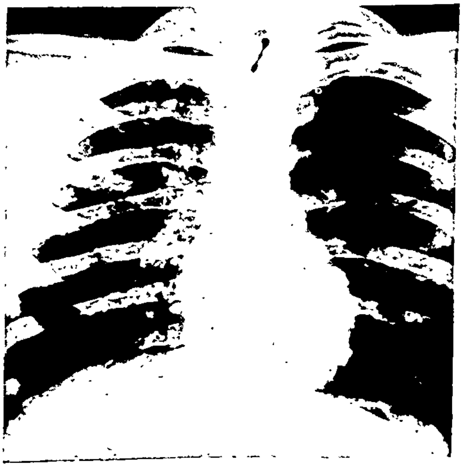

Fig. 5.—(Case 2.) Preoperative roentgenogram.

As an incidental finding it was noted that twenty-eight patients, or 17 per cent, had been treated by artificial pneumothorax, an average of 5.5 years with reformation of cavities after re-expansion (Table V).

## COMMENT

This study has been undertaken in an attempt to evaluate the effectivity and validity of a limited thoracoplasty done in conjunction with a large extrafascial apicolysis in the treatment of apical tuberculosis with cavitation. These

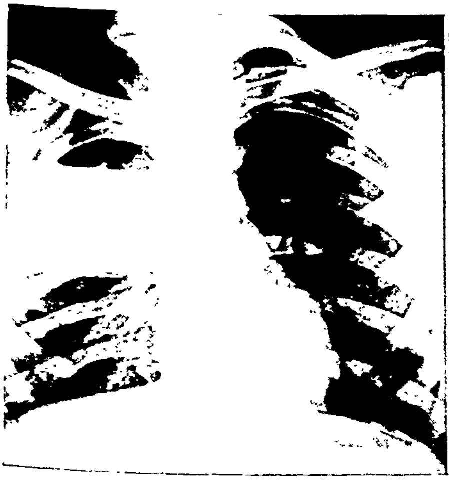

Fig. 6.

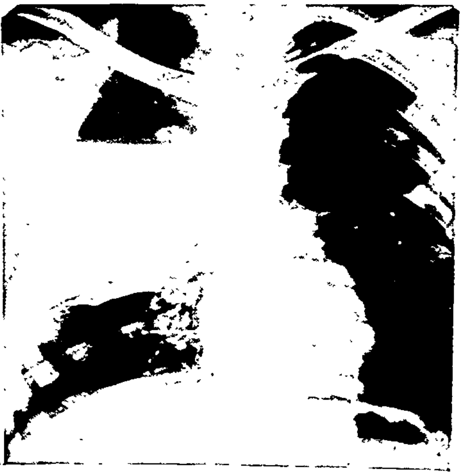

Fig. 7.

Fig. 6.—Case 2 after first stage and one air filling.
Fig. 7.—Case 2 after second stage and additional filling.

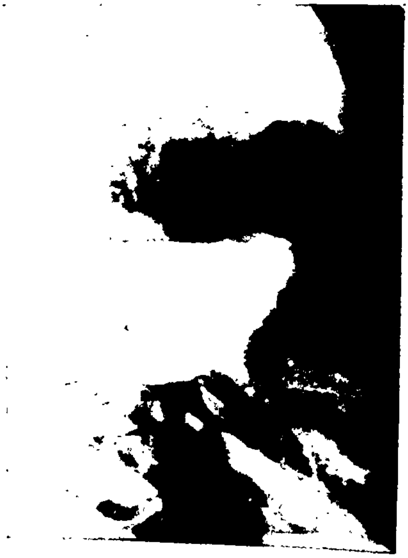

Fig. 8.—Case 2 after second stage, lateral view.

Table IV. Early and Late Results Correlated with Cavity Size
3, 4, and 5 Rib Thoracoplasties

| CAVITY SIZE | NO. | INF. | MORT. LESS THAN 2 MO. | MORT. LATER | CAV. - TB. - | CAV. + TB. + |
|---|---|---|---|---|---|---|
| I<br>0-2.5 cm. | 142 | 8 | 1 | 5 | 129<br>90.8 per cent | 7<br>4.9 per cent |
| II<br>2.5-4 cm. | 18 | - | - | 1 | 13<br>72.2 per cent | 4<br>22.2 per cent |
| III<br>4-5 cm. | 3 | - | - | - | 3<br>100 per cent | - |
| Total | 163 | 8 | 1 | 6 | 145<br>88.9 per cent | 11<br>7.6 per cent |

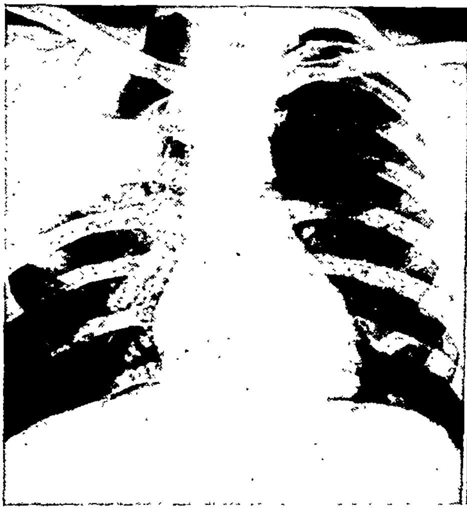

Fig. 9.

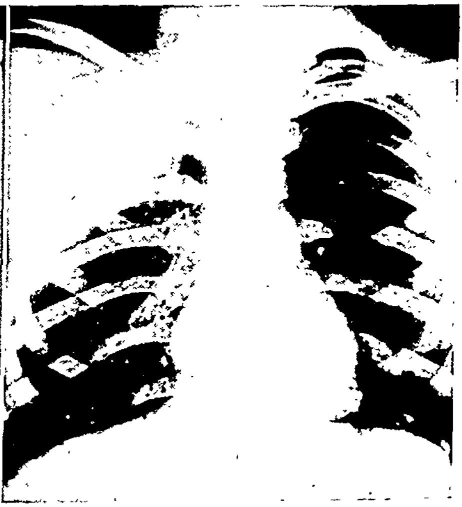

Fig. 10.

Fig. 9.—Case 2 after 3 months; air filling discontinued.
Fig. 10.—Case 2, four months after air filling was discontinued.

Table V. Number of Years Between the Institution of Pneumothorax and Operation. Residual Cavity after A-P.

| | | |
|---|---|---|
| 5 ribs | 19 | Av. Years between A-P and operation<br>5.5 |
| 4 ribs | 8 | |
| 3 ribs | 1 | |
| Total | 28—17 per cent | |

results illustrate what can be achieved with a minimum of outward, and more important, inward deformity. The diseased portion of the lung with its cavity or cavities is permanently collapsed, allowing a minimal loss of function of the remaining unaffected lung. This has been achieved with a low mortality both postoperative and late. Planigraphic studies have been carried out in the majority of cases both before and after operation (Figs. 11 and 12). Recently planigrams have been taken routinely pre- and postoperatively to study the relationship of the drainage bronchus to the ultimate result. In several of the cases of residual cavity open drainage bronchi can be demonstrated.

To achieve success with such a limited rib resection, especially in the four-rib one-stage operations, an extensive and exact extrafascial apicolysis is of the utmost importance. Of almost equal importance is the vigilant filling, preferably with air, of the apicolysis space until regenerated ribs are fully formed.

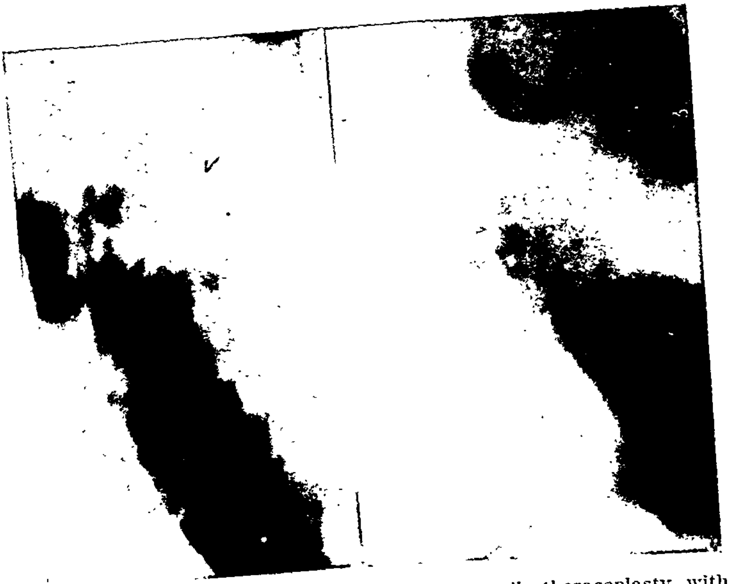

Fig. 11.—Planigrams before and ten days after a five-rib thoracoplasty with extrafascial apicolysis.

Fig. 12.—Planigrams before and ten days after a five-rib thoracoplasty with extrafascial apicolysis.

## CASE REPORTS

CASE 1.—This 42-year-old white woman developed pleurisy in 1926. She was treated by a right pneumothorax from 1936 to 1940. In December, 1946, she became weak, began to cough, and a chest roentgenogram revealed bilateral apical infiltration with cavitation (Fig. 3). Pneumothorax was unsuccessful. Sputum was positive on direct smear, vital capacity was 2,500 c.c., and there was excellent diaphragm movement on both sides. Because of her good general condition and the small cavities located well up in the apices, it was decided to perform bilateral four-rib thoracoplasties with extrafascial apicolysis. The left side was done first. The right side was done after a four-month interval (Fig. 4). Four months later the sputum was negative on smear but positive a single instance in a culture from gastric contents. Planigrams were negative for residual cavity.

CASE 2.—This 18-year-old man developed tuberculosis in 1947. He was admitted to Oslo Municipal Hospital four months later with bilateral infiltration and right upper lobe cavitation. The sputum was strongly positive on direct smear. Pneumothorax was unsuccessful on the left and contraselective. His vital capacity was 3,600 c.c., and diaphragmatic movement was excellent. The daily sputum varied from 10 to 30 c.c. A five-rib thoracoplasty with extrafascial apicolysis was therefore performed in two stages in conjunction with air filling. Two years after the final stage he was well and working with negative sputum cultures and no planigraphic evidence of residual cavitation (Figs. 5 to 10).

## SUMMARY

One hundred seventy-four cases of tuberculosis of the lungs treated by thoracoplasty with extrafascial apicolysis involving five or less ribs with a total of 261 stages of operation are reviewed. The results are presented and briefly discussed.

## REFERENCE

1. Semb, Carl: Thoracoplasty with Extrafascial Apicolysis, Acta chir. Scandinav. 76: 1-85, 1935. Supp. 37 (art. 2).

# PULMONARY RESECTION FOR TUBERCULOSIS

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PULMONARY resections for tuberculosis have been reported over a period of half a century, but it is only during the past decade that resection has become recognized as an approved therapeutic measure, and that any large series of cases has been reported. Sporadic instances of resections were performed on Olive View patients before the introduction of streptomycin, and in a few instances the results were gratifying, but the incidence of bronchopleural fistula and empyema, and the high operative mortality in the cases operated by the old tourniquet method and without the aid of streptomycin, resulted in the reservation of this procedure for cases in which nothing else could be done. During the past four years, however, resection has been increasing in popularity here, as elsewhere, and is now recognized as an established procedure.

This paper presents a critical analysis of all cases of pulmonary resection for tuberculosis performed by the individual ligation technique at Olive View Sanatorium through Dec. 31, 1950. The series comprises eighty-four consecutive cases of pulmonary resection, of which one operation was performed in 1947, four in 1948, thirty-six in 1949, and forty-three in 1950. Complete follow-up data were obtained as of Nov. 1, 1951, in all but two cases. The last follow-up in one of the cases was one year after the surgery in June, 1950. The other follow-up was made in August, 1950, four months after surgery. In these two cases we are using the information as of that date.

In order to evaluate the resections, the results are divided into five categories: good, satisfactory, fair, poor, and deaths. The first three groups are further divided into working and not working classifications. Criteria used for establishing the groups are as follows:

*Good:* Cases that have done well and have remained well. All sputum concentrates, cultures, and gastric cultures have been negative since surgery. There were no spreads, no postoperative complications, no bronchopleural fistulas except in one case in which the bronchial stump leaked immediately and was repaired within twenty-four hours. This case is being included since it was obviously a surgical defect and had no connection with the result, so far as tuberculosis is concerned. In this group there are twenty-nine cases; thirteen of whom are now working, five full time.

*Satisfactory:* In this group are placed the cases who are as well now as are the cases in the *good* group, but have had, since surgery, one or more of the following: (a) positive sputum or gastric culture, now negative, (b) a spread that has cleared, (c) bronchopleural fistula, now closed, and (d) other postoperative complications from which there has been complete recovery. In this group are twenty-five cases, seven of whom are now working, one full time.

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TABLE I. RESULTS OF PULMONARY RESECTION FOR TUBERCULOSIS

| | GOOD WORKING | | SATISFACTORY WORKING | | FAIR WORKING | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | YES | NO | YES | NO | YES | NO | POOR | DIED | TOTALS |
| Sex and Age, Males 10-29 | 4 | 2 | 0 | 2 | 0 | 2 | 1 | 1 | 12 |
| 30-39 | 2 | 0 | 4 | 3 | 1 | 0 | 0 | 4 | 14 |
| 40-49 | 3 | 4 | 1 | 1 | 0 | 1 | 1 | 0 | 11 |
| 50 and over | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 4 |
| Totals | 9 | 7 | 5 | 6 | 1 | 4 | 3 | 6 | 41 |
| Females 10-29 | 2 | 1 | 2 | 6 | 1 | 2 | 2 | 1 | 17 |
| 30-39 | 2 | 3 | 0 | 1 | 1 | 3 | 3 | 1 | 14 |
| 40-49 | 0 | 4 | 0 | 3 | 0 | 1 | 0 | 0 | 8 |
| 50 and over | 0 | 1 | 0 | 2 | 0 | 0 | 0 | 1 | 4 |
| Totals | 4 | 9 | 2 | 12 | 2 | 6 | 5 | 3 | 43 |
| Race White | 3 | 12 | 3 | 5 | 2 | 3 | 1 | 3 | 32 |
| Mexican | 8 | 4 | 2 | 8 | 1 | 5 | 4 | 3 | 35 |
| Negro | 1 | 0 | 1 | 3 | 0 | 1 | 1 | 2 | 9 |
| Other | 1 | 0 | 1 | 2 | 0 | 1 | 2 | 1 | 8 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Patients working, 1-4 hr. Men | 3 | | 3 | | 1 | | | | 7 |
| Women | 4 | | 2 | | 0 | | | | 6 |
| 5-7 hr. Men | 1 | | 1 | | 0 | | | | 2 |
| Women | 0 | | 0 | | 2 | | | | 2 |
| 8-9 hr. Men | 5 | | 1 | | 0 | | | | 6 |
| Women | 0 | | 0 | | 0 | | | | 0 |
| Totals | 13 | 0 | 7 | 0 | 3 | 0 | 0 | 0 | 23 |
| Disease: Unilateral | 0 | 2 | 1 | 1 | 0 | 0 | 0 | 0 | 4 |
| Unilateral with cavity | 1 | 2 | 0 | 2 | 0 | 1 | 0 | 0 | 6 |
| Bilateral | 8 | 6 | 2 | 5 | 1 | 3 | 3 | 5 | 33 |
| Bilateral with unilateral cavity | 4 | 6 | 4 | 10 | 2 | 6 | 5 | 4 | 41 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Cavity size: under 2 cm. | 5 | 5 | 1 | 5 | 0 | 3 | 3 | 0 | 22 |
| 2 cm. and over | 0 | 2 | 4 | 6 | 2 | 4 | 1 | 4 | 23 |
| Totals | 5 | 7 | 5 | 11 | 2 | 7 | 4 | 4 | 45 |
| Cavity character: Slit | 2 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 5 |
| Bronchiectatic | 1 | 2 | 0 | 2 | 0 | 1 | 1 | 0 | 7 |
| Irregular | 0 | 1 | 1 | 2 | 1 | 3 | 0 | 1 | 9 |
| Round | 0 | 1 | 2 | 2 | 0 | 1 | 1 | 1 | 8 |
| Totals | 3 | 6 | 4 | 6 | 1 | 5 | 2 | 2 | 29 |
| Cavity location: Upper lobe | 4 | 6 | 3 | 11 | 1 | 4 | 4 | 4 | 37 |
| Lower lobe | 1 | 1 | 2 | 1 | 1 | 3 | 0 | 0 | 9 |
| Totals | 5 | 7 | 5 | 12 | 2 | 7 | 4 | 4 | 46 |
| Duration of disease: 0-1 yr. | 1 | 3 | 1 | 1 | 0 | 0 | 2 | 1 | 9 |
| 2-4 yr. | 3 | 1 | 2 | 7 | 0 | 3 | 2 | 2 | 20 |
| 5-9 yr. | 5 | 9 | 2 | 8 | 2 | 4 | 3 | 2 | 35 |
| 10 and over | 2 | 3 | 1 | 1 | 1 | 1 | 0 | 2 | 11 |
| Totals | 11 | 16 | 6 | 17 | 3 | 8 | 7 | 7 | 75 |
| Preoperative sputum, Positive Conc. | 2 | 6 | 3 | 8 | 2 | 5 | 3 | 3 | 32 |
| Intermittent Pos. Conc. | 1 | 4 | 1 | 3 | 0 | 1 | 0 | 1 | 11 |
| Occasional Pos. Conc. | 3 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 6 |
| Neg. Conc.—Pos. Culture | 2 | 1 | 1 | 4 | 0 | 3 | 3 | 3 | 17 |
| Neg. Conc. Inter. Pos. Culture | 3 | 2 | 1 | 2 | 0 | 1 | 1 | 1 | 11 |
| Neg. Conc. Occas. Pos. Culture | 0 | 2 | 0 | 1 | 1 | 0 | 0 | 0 | 4 |
| Neg. on Conc., Cult. and Gastric | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Previous thoracoplasty: 3 ribs | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 5 ribs | 1 | 4 | 2 | 5 | 1 | 1 | 2 | 0 | 16 |
| 6 ribs | 0 | 3 | 1 | 1 | 1 | 0 | 2 | 1 | 9 |
| 7 ribs | 5 | 5 | 1 | 6 | 1 | 3 | 4 | 3 | 28 |
| 8 ribs | 2 | 0 | 0 | 2 | 0 | 2 | 0 | 2 | 8 |
| 9 ribs | 1 | 1 | 1 | 1 | 0 | 1 | 0 | 1 | 6 |
| Totals | 9 | 13 | 5 | 16 | 3 | 7 | 8 | 7 | 68 |

Table I.—Cont'd

| | GOOD WORKING | | SATISFACTORY WORKING | | FAIR WORKING | | POOR | DIED | TOTALS |
|---|---|---|---|---|---|---|---|---|---|
| | YES | NO | YES | NO | YES | NO | | | |
| Types of resection: primary | 4 | 3 | 2 | 2 | 0 | 3 | 0 | 2 | 16 |
| after homolateral thoracoplasty | 4 | 11 | 4 | 14 | 3 | 6 | 6 | 4 | 52 |
| after bilateral thoracoplasty | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| after contralateral extrapleural pneumothorax and homolateral thoracoplasty | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| after revision thoracoplasty | 3 | 0 | 0 | 1 | 0 | 1 | 1 | 1 | 7 |
| after lucite pack and thoracoplasty | 2 | 2 | 1 | 0 | 0 | 0 | 1 | 1 | 7 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Streptomycin 5-39 grams | 9 | 7 | 3 | 13 | 2 | 5 | 2 | 2 | 43 |
| 40 or more | 3 | 9 | 4 | 5 | 1 | 5 | 5 | 6 | 38 |
| No streptomycin | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 3 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Preoperative bronchoscopy | | | | | | | | | |
| No discharge | 4 | 4 | 0 | 6 | 1 | 3 | 2 | 3 | 23 |
| Secretions, homolateral | 3 | 6 | 1 | 7 | 0 | 2 | 2 | 2 | 23 |
| Secretions, contralateral side | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 22 |
| Secretions, both sides | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Stenosis homolateral | 1 | 5 | 1 | 4 | 0 | 4 | 2 | 1 | 18 |
| Totals | 8 | 17 | 2 | 18 | 1 | 9 | 6 | 6 | 67 |
| Streptomycin resistant | 0 | 3 | 0 | 3 | 0 | 1 | 5 | 4 | 16 |
| Not streptomycin resistant | 13 | 13 | 7 | 15 | 3 | 8 | 4 | 5 | 68 |
| Totals | 13 | 16 | 7 | 18 | 3 | 9 | 9 | 9 | 84 |
| Previous tracheobronchial disease | 4 | 6 | 2 | 2 | 0 | 3 | 6 | 0 | 23 |
| No previous tracheobronchial disease | 9 | 10 | 5 | 16 | 3 | 6 | 3 | 9 | 61 |
| Totals | 13 | 16 | 7 | 18 | 3 | 9 | 9 | 9 | 84 |
| Operation: | | | | | | | | | |
| Pneumonectomy, left | 4 | 6 | 2 | 6 | 0 | 3 | 3 | 4 | 28 |
| Pneumonectomy, right | 3 | 7 | 4 | 3 | 1 | 4 | 2 | 4 | 28 |
| Lobectomy, left upper | 1 | 2 | 0 | 5 | 0 | 1 | 0 | 1 | 10 |
| Lobectomy, right upper | 3 | 0 | 0 | 3 | 0 | 1 | 2 | 0 | 9 |
| Lobectomy, left lower & lingula | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Lobectomy, left lower | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| Lobectomy, right lower & middle | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| Lobectomy, right upper & middle | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 3 |
| Lobectomy, left upper and sup. segment of lower | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 2 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Operating time: 3-3:59 hr. | 0 | 2 | 0 | 3 | 2 | 0 | 1 | 0 | 8 |
| 4-4:59 hr. | 2 | 5 | 0 | 4 | 0 | 3 | 3 | 1 | 18 |
| 5-5:59 hr. | 3 | 4 | 4 | 3 | 0 | 4 | 1 | 1 | 20 |
| 6-6:59 hr. | 7 | 4 | 2 | 3 | 1 | 1 | 1 | 4 | 23 |
| 7-7:59 hr. | 1 | 1 | 0 | 4 | 0 | 0 | 1 | 2 | 9 |
| 8-8:59 hr. | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 2 |
| 9-9:59 hr. | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| Totals | 13 | 16 | 6 | 17 | 3 | 10 | 8 | 8 | 81 |
| Operative and/or anesthetic difficulty | 4 | 2 | 1 | 2 | 1 | 3 | 4 | 6 | 23 |
| No especial difficulty | 9 | 14 | 6 | 16 | 2 | 7 | 4 | 3 | 61 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Blood loss: Under 1000 cc. | 1 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 3 |
| 1000 to 1999 cc. | 3 | 2 | 2 | 5 | 0 | 3 | 1 | 1 | 17 |
| 2000 to 2999 cc. | 6 | 8 | 3 | 5 | 1 | 4 | 5 | 2 | 34 |
| 3000 to 3999 cc. | 3 | 4 | 1 | 1 | 1 | 2 | 2 | 3 | 17 |
| 4000 to 4999 cc. | 0 | 2 | 0 | 2 | 1 | 0 | 0 | 0 | 5 |
| 5000 to 5999 cc. | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 6000 to 6999 cc. | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 |

TABLE I.—CONT'D

| | GOOD WORKING | | SATISFACTORY WORKING | | FAIR WORKING | | POOR | DIED | TOTALS |
|---|---|---|---|---|---|---|---|---|---|
| | YES | NO | YES | NO | YES | NO | | | |
| 7,000 to 7,999 c.c. | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| Totals | 13 | 16 | 6 | 16 | 3 | 9 | 9 | 8 | 80 |
| Preoperative hemoglobin: 64-79 | 5 | 2 | 1 | 4 | 1 | 6 | 1 | 5 | 25 |
| 80-89 | 5 | 9 | 2 | 10 | 1 | 1 | 6 | 0 | 34 |
| 90-100 | 3 | 4 | 3 | 2 | 1 | 2 | 1 | 2 | 18 |
| Totals | 13 | 15 | 6 | 16 | 3 | 9 | 8 | 7 | 77 |
| Postoperative fever: (above 98.6° F.) 1- 5 days | 0 | 0 | 0 | 1 | 1 | 2 | 0 | 1 | 5 |
| 6-10 days | 4 | 4 | 4 | 5 | 2 | 3 | 1 | 1 | 24 |
| 11-20 days | 4 | 5 | 1 | 8 | 0 | 2 | 1 | 0 | 21 |
| 21-30 days | 3 | 3 | 0 | 2 | 0 | 0 | 0 | 3 | 11 |
| 31-40 days | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 | 3 |
| 41 and over | 1 | 3 | 0 | 1 | 0 | 0 | 0 | 1 | 6 |
| Totals | 12 | 15 | 6 | 18 | 3 | 7 | 2 | 7 | 70 |
| Spreads one, first postoperative month | 0 | 0 | 2 | 10 | 0 | 1 | 1 | 3 | 17 |
| Spreads one, after first postoperative month | 0 | 0 | 1 | 2 | 0 | 1 | 6 | 1 | 11 |
| Spreads two or more, first month and later | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 2 | 5 |
| No spreads | 13 | 16 | 4 | 6 | 3 | 6 | 0 | 3 | 51 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 9 | 84 |
| Bronchopleural fistula: first day | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Bronchopleural fistula within 1 month | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 2 |
| Bronchopleural fistula after first month | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| Totals | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 2 | 4 |
| Postoperative sputums: | | | | | | | | | |
| All conc., cult., & gast. neg. | 12 | 14 | 3 | 13 | 0 | 3 | 1 | 0 | 46 |
| Neg. conc., cult. No gast. rep. | 1 | 2 | 0 | 1 | 0 | 0 | 0 | 2 | 6 |
| All conc., cult., gast. neg. except preceded by 1 pos. cult. | 0 | 0 | 3 | 2 | 0 | 1 | 0 | 0 | 6 |
| Neg. on conc., cult., gast. except preceded by occ. pos. culture | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 3 |
| Neg. on conc., cult., gast. except preceded by occ. pos. conc. | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 2 | 3 |
| Neg. conc.; occ pos. cult. or gastric | 0 | 0 | 0 | 0 | 2 | 0 | 1 | 0 | 3 |
| Neg. on conc., pos. on cult. | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 2 |
| Neg. conc., occ. pos. culture | 0 | 0 | 0 | 0 | 1 | 4 | 1 | 0 | 6 |
| Occasional pos. concentrate | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 2 | 5 |
| Regularly positive concen. | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 |
| Totals | 13 | 16 | 7 | 18 | 3 | 10 | 8 | 7 | 82 |

| | TOTAL NUMBER OF CASES | GOOD AND SATISFACTORY | PERCENTAGE OF CASES WITH GOOD AND SATISFACTORY RESULTS |
|---|---|---|---|
| Males: Age 10-29 | 12 | 8 | 67 |
| 30-39 | 14 | 9 | 64 |
| 40 and over | 15 | 10 | 67 |
| Females: 10-29 | 17 | 11 | 65 |
| 30-39 | 14 | 6 | 43 |
| 40 and over | 12 | 10 | 83 |
| Race: White | 32 | 23 | 72 |
| Mexican | 35 | 22 | 63 |
| Negro | 9 | 5 | 55 |
| Other | 8 | 4 | 50 |
| Disease: Unilateral | 4 | 4 | 100 |
| Unilateral with cavity | 6 | 5 | 83 |
| Bilateral | 33 | 21 | 64 |
| Bilateral with unilateral cavity | 41 | 24 | 58 |

TABLE I.—CONT'D

| | TOTAL NUMBER OF CASES | GOOD AND SATISFACTORY | PERCENTAGE OF CASES WITH GOOD AND SATISFACTORY RESULTS |
|---|---|---|---|
| Cavity size: Under 2 cm. | 22 | 16 | 73 |
| Over 2 cm. | 23 | 12 | 52 |
| Cavity character: Slit | 5 | 5 | 100 |
| Bronchiectatic | 7 | 5 | 71 |
| Irregular | 9 | 4 | 44 |
| Round | 8 | 5 | 62 |
| Cavity location: Upper lobe | 37 | 24 | 65 |
| Lower lobe | 9 | 5 | 55 |
| Duration of disease: 0-1 yr. | 9 | 6 | 67 |
| 2-4 yr. | 20 | 13 | 65 |
| 5-9 yr. | 35 | 24 | 68 |
| 10 and over | 11 | 7 | 64 |
| Preoperative sputum: Pos. Conc. | 49 | 34 | 69 |
| Preoperative neg. concentrate; pos. culture | 32 | 19 | 60 |
| Preoperative neg. concentrate; cult.; & gast. | 3 | 3 | 100 |
| Previous thoracoplasty: | | | |
| 5 ribs or less | 17 | 13 | 76 |
| 6 ribs or more | 51 | 30 | 59 |
| Type of Resection: Primary | 16 | 11 | 69 |
| Type: post thoracoplasty | 68 | 43 | 63 |
| Type primary resection: | | | |
| No thoracoplasty | 8 | 7 | 88 |
| Postresection thoracoplasty | 8 | 4 | 50 |
| Streptomycin: 5-39 Gm. | 43 | 32 | 72 |
| 40 or more Gm. | 38 | 21 | 55 |
| Preop. bronchoscopy: no secretions | 23 | 14 | 61 |
| Preop. bronchoscopy: homolateral secretions | 23 | 17 | 74 |
| Preop. bronchoscopy: homolateral stenosis | 18 | 11 | 61 |
| Streptomycin resistant | 16 | 6 | 38 |
| Streptomycin not resistant | 68 | 48 | 71 |
| Previous tracheobronchial disease | 23 | 14 | 61 |
| No previous tracheobronchial disease | 61 | 40 | 66 |
| Operation: Pneumonectomy | 56 | 35 | 63 |
| Lobectomy | 28 | 19 | 68 |
| Operating time: Under 7 hours | 69 | 46 | 67 |
| Over 7 hours | 12 | 6 | 50 |
| Operative and/or anes. difficulty | 23 | 9 | 39 |
| No operative and/or anes. difficulty | 61 | 45 | 74 |
| Blood Loss: Under 3,000 c.c. | 54 | 37 | 69 |
| Over 3,000 c.c. | 26 | 14 | 54 |
| Preoperative hemoglobin under | | | |
| 80 per cent | 25 | 12 | 48 |
| over 80 per cent | 52 | 38 | 73 |
| Postoperative febrile course: | | | |
| 1-10 days | 29 | 18 | 62 |
| over 10 days | 41 | 33 | 80 |
| Spreads: All types | 33 | 15 | 45 |
| No spreads | 51 | 39 | 77 |
| Bronchopleural fistulas | 4 | 2 | 50 |

*Fair:* Some cases show an occasional positive sputum or gastric culture, yet remain fairly well. Others are completely negative on concentrate culture and gastric culture, but show slight residual infiltration; if a bronchopleural fistula was present, it is now closed. In this group there are thirteen cases, three of whom are now working, none full time.

*Poor:* These cases are not doing well. They show increase in disease, positive sputum, or bronchopleural fistula. Eight cases are in this group.

*Deaths:* There are nine deaths in this series, an over-all death rate of 10.7 per cent. There were two deaths within three days, each directly due to the surgery with an immediate surgical mortality rate of 2.4 per cent. The other deaths all occurred after four months.

The association of various factors with the results observed from one to four years after resection is shown in table form. From these figures the percentages of favorable results were calculated and significant relationships noted.

*Review of Results.—Sex:* Essentially no difference. *Age:* Little influence except in women over 40 years, who had best results. *Race:* Best in white race, next best in Mexicans, poorest in Japanese and Negroes. *Disease:* Unilateral disease far better results than bilateral disease. Cases with cavity less satisfactory results than those without cavity; but unilateral cavity cases did better than those with bilateral disease without cavity. *Cavity size:* Better results with small cavities, under 2 centimeters. *Cavity character:* Best results with slit and bronchiectatic cavities. All cases of slit cavity had a good result. *Cavity location:* No significant differences. *Duration of disease:* No significant differences. *Preoperative sputum:* Best results in the three cases who had negative sputum and gastric cultures. Cases with positive concentrates did better than those with negative concentrates and positive cultures.

The indications for resection in the three cases who were negative: (1) surgical emergency for hemoptysis. Previous four stages of thoracoplasty and revision; negative sputum since then. Left pneumonectomy. (2) Cavity in apex of left lung, seen on x-ray. Previous six rib thoracoplasty, negative sputum and gastric cultures since then. Left upper lobectomy. (3) Unexpandable diseased upper lobe. Pleural effusion and atelectatic lung with residual pneumothorax. Sputum and gastric cultures negative. Decorticated but upper lobe failed to expand. Left upper lobectomy.

*Previous thoracoplasty:* Best results with five ribs or less. *Type of resection:* Results best in resections not associated with thoracoplasty; but the primary resections followed by thoracoplasty did worse than those whose resection was preceded by thoracoplasty. *Streptomycin resistant:* Results almost twice as good in the cases who were not resistant to streptomycin, either partially or entirely. In the entire series this was one of the most outstanding findings.

*Previous tracheobronchial disease:* Results not significantly better in the cases who did not have previous disease. *Operation performed:* Results slightly better with lobectomy than with pneumonectomy, but difference not statistically significant. *Duration of operation:* Cases who required under seven hours did slightly better than those over seven hours. *Operative and/or anesthetic difficulties:* Results almost twice as good in the cases without difficulty. Most frequently encountered was difficulty in freeing the lung due to adhesions and adherence to the chest wall. *Blood loss:* Cases with blood loss under 3,000 c.c. did far better than those in which the loss was greater. Blood loss was generally greater in the cases in which difficulty in freeing the lung was present.

*Preoperative hemoglobin:* Cases with over 80 per cent did far better than those under 80 per cent. *Preoperative febrile course:* Until a case leveled off at 98.6° F. or under, that case was listed as having fever. Cases with febrile course over ten days did better than those under ten days. *Spreads:* Cases without spreads did far better than those with spreads. *Bronchopleural fistulas:* Two of the four cases with fistula had good results. One was immediately recognized so that closure was done within twenty-four hours; the other showed up four days after surgery and closed spontaneously within another week or so.

Significant hazards to obtaining a good result were:

1. Operative or anesthetic difficulties.
2. Resistance to streptomycin.
3. Spreads.
4. Bilateral disease.
5. Round or irregular cavity.
6. Cavity over 2 cm. in size.
7. Regular positive cultures or concentrates.
8. Preoperative hemoglobin under 80 per cent.
9. Blood loss over 3,000 c.c.
10. Previous six or more rib thoracoplasty.

*Spreads:* During the first postoperative month there were seventeen cases which had a spread. Any increase in pulmonary density was counted as a spread, although several cases so classified may have been due to atelectasis or hemorrhage into the bronchioles, since the x-ray shadow cleared rather rapidly.

*Outcome of the seventeen spreads that occurred during the first postoperative month:* Twelve cases showed complete clearing on radiographic examination and became completely negative on sputum and gastric cultures. Those cases were included in the satisfactory category. Three cases were classified as fair results; of these, one had a slight residual infiltration but became negative on sputum and gastric examinations. The second case had additional later spreads, but became negative on concentrate culture and gastric, but had residual soft infiltration. The third case had a later spread which completely cleared on x-ray examination, but the cultures were intermittently positive. Two cases were classified as poor; one developed a cavity and was intermittently positive on concentrate; the other had a later spread and was regularly positive on concentrate.

Of the thirty-three patients who had spreads, at any time following operation, one who developed soft infiltration and two who formed cavities died. Of the thirty surviving patients who had spreads, regression, fibrosis, or calcification has taken place in nineteen, but seven still have residual soft infiltration, and four others show definite cavitation.

*Survivals:* Of the seventy-five patients surviving in this series, sixty-four have no roentgenographic evidence of any soft infiltration, cavitation, or empyema, and are thus satisfactory results, from an x-ray standpoint. Four of

*Poor:* These cases are not doing well. They show increase in disease, positive sputum, or bronchopleural fistula. Eight cases are in this group.

*Deaths:* There are nine deaths in this series, an over-all death rate of 10.7 per cent. There were two deaths within three days, each directly due to the surgery with an immediate surgical mortality rate of 2.4 per cent. The other deaths all occurred after four months.

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twenty-three are already working. Less satisfactory results were obtained in the Negro race, patients with larger or tension cavities, and in those already resistant to streptomycin. Poor results were also noted in patients who developed postoperative spreads and in those who required more chemotherapy or post-resection thoracoplasty.

Pulmonary resection is a valuable therapeutic measure in selected cases of pulmonary tuberculosis, especially for lesions which have not been controlled by thoracoplasty. Pulmonary resection should *not* be lightly undertaken. Careful selection and preparation of patients, meticulous surgical technique and painstaking postoperative care are required to minimize its dangers and insure maximum results.

the seventy-five patients have positive concentrates, and eleven have positive cultures. Thus, 20 per cent still have positive sputums. Fifteen others were positive following resection, but are now negative. In addition to the obvious improvements in physical findings, roentgenogram, and laboratory data, many patients had marked lessening or disappearance of symptoms such as wheezing, anorexia, night sweats, coughing, and raising of sputum.

*Review of Deaths.*—1. White man, aged 36 years, streptomycin resistant, positive sputum, developed tuberculous meningitis four months after left pneumonectomy after previous thoracoplasty. He died three months later.

2. A Mexican man, aged 50 years, had positive sputum. He had a left pneumonectomy after a previous thoracoplasty. The operation required more than seven hours. The patient left the operating room in poor condition and died one hour later.

3. A Mexican woman, aged 19 years, had positive sputum and contralateral oleothorax. She had an extensive bronchopneumonic spread after a right pneumonectomy after a previous thoracoplasty. She died three days later.

4. A Mexican man, aged 19 years, had a bronchopleural fistula. He had a right pneumonectomy after a previous thoracoplasty. He subsequently had a spread and hydropneumothorax. He had a skin flap drainage; and he died seven months after resection.

5. A white woman, aged 59 years, had positive sputum. She ran an intermittent febrile course after a primary right pneumonectomy. She had a postresection thoracoplasty, and later had exploratory thoracotomy. She died eleven months after resection; autopsy showed widespread tuberculosis and right empyema.

6. A Negro man, aged 37 years, had a positive sputum. He had a left pneumonectomy after a previous thoracoplasty. He developed acute cor pulmonale and auricular fibrillation and died four months later.

7. A white man, aged 32 years, had a positive sputum. He died two years after a right pneumonectomy after a previous thoracoplasty. He had previously been discharged from Olive View; cause of death unknown.

8. A Negro man, aged 35 years, had a positive sputum. He had an early spread after left pneumonectomy after a previous thoracoplasty which completely regressed eighteen months after surgery. He was discharged and died on the outside two and three-fourths years after resection; cause of death unknown.

9. A Japanese woman, aged 31 years, had a positive sputum. She had a spread with cavity and hemoptysis, and died ten months after primary right pneumonectomy.

## SUMMARY AND CONCLUSIONS

Of eighty-four resections for pulmonary tuberculosis at Olive View between 1947 and 1950, fifty-four gave satisfactory results, twenty-one unsatisfactory, and nine died. There was an immediate surgical mortality rate of 2.4 per cent, with an over-all death rate of 10.7 per cent. Follow-up of the seventy-five survivals one to four years later shows sixty with negative sputum, of whom layers of rubber cement.[*] Excess rubber at the base of the sixth finger is removed: the tips of the added finger and the index finger are cut off and small cuffs are turned and cemented (Fig. 1, *B*). The glove is placed in an oven at 200° F. for half an hour, after which the test tube is removed and the glove is tested for leaks. It is then packaged and autoclaved in the usual manner.

The six-fingered glove is employed in commissurotomy after the purse-string ligatures are placed in the base of the auricular appendage and the tip of the appendage is amputated distal to a noncrushing clamp. The glove is put on over the standard operating glove. The guillotine knife is moistened in saline and slipped through the added "sixth" finger and out the index finger. The end of the sixth finger which has become an instrument tunnel is then closed tightly with two turns of a heavy silk ligature (Fig. 1, *C*). With the forefinger and instrument in the auricle, blood flows into the extra finger and space between the two gloves, but can go no further. Occasionally with very high auricular pressures it has been necessary to place a tape ligature around the operator's wrist to close that avenue of escape.

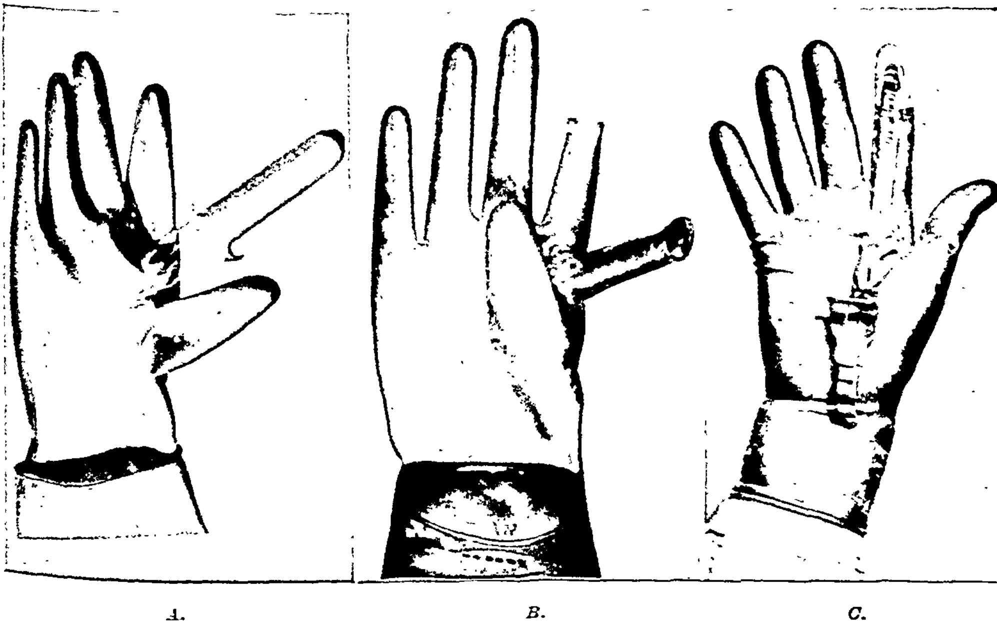

Fig. 1.—Six-fingered glove for use in mitral commissurotomy: *A*, Method of adding sixth finger to glove. *B*, Completed glove. *C*, Use of the glove with the new model "push" type of Bailey-Glover-O'Neill commissurotomy guillotine. Note that the glove is applied over the usual surgical glove and the guillotine knife is inserted through the sixth finger and out the index finger. A ligature fastens the sixth finger securely to the knife.

## SUMMARY

A six-fingered glove for the further prevention of hemorrhage during mitral commissurotomy has been described.

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[*] John Bunn Corporation, Buffalo, N. Y.

# SIX-FINGERED GLOVE FOR MITRAL COMMISSUROTOMY

EDWARD R. MUNNELL, M.D.
DETROIT, MICH.

OPERATIONS for mitral stenosis involving the insertion of a forefinger into the left auricle by way of its appendage have resulted in dramatic improvement in many patients.[1-4] While the finger is in the heart, hemostasis is usually easily obtained by the tightening of one or more purse-string ligatures previously placed around the base of the appendage. However, if attempts to open the valve by blunt dissection or "finger fracture" are unsuccessful, it becomes necessary to introduce some type of cutting instrument along the finger. With the high auricular pressure encountered in mitral stenosis, the presence of the instrument between the finger and the appendage wall may permit the escape of considerable amounts of blood. Bailey, Glover, and O'Neill described a technique to minimize this bleeding. A second glove was placed on the operator's hand, and the commissurotomy knife was passed through a slit in the palm and out through the amputated tip of the forefinger of the glove. Harken has used a part of a finger cot for this purpose. In fifteen mitral commissurotomies, Dr. Conrad Lam employed the two-glove technique and noted that there was frequently troublesome bleeding from the hole in the outer glove from which the knife emerged. In one patient there was post-commissurotomy hypotension apparently due to excessive blood loss, and in others it was necessary to make hasty decisions as to the adequacy of the incisions into the commissures.

The six-fingered glove was designed to obviate this remaining source of hemorrhage. The use and advantages of this glove have been mentioned in a previous paper.[5] It has now been used by Dr. Lam in twenty additional cases. It has reduced blood loss to negligible amounts, and has enabled the surgeon to explore and incise the stenotic mitral valve without urgency. The purpose of this communication is to describe the construction of the glove for the benefit of others who may feel the need of it.

The third finger of a surgical glove is removed at its base, taking liberal margins from the palmar and dorsal surfaces of the glove (Fig. 1, *A*). This is then placed over a test tube which the rubber finger will fit snugly. A star is then cut in the recipient glove on the palmar surface approximately halfway between the thenar eminence and the index finger. The finger to be added and test tube are inserted through the star to the desired position. The rubber in the region of apposition is cleaned with benzene and cemented with several

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